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ABSTRACT

A variety of socio-economic criteria are suggested for the choice of how best to utilize energy resources. One of the most significant of these criteria is the prediction and evaluation of existing and potential human health effects of recovery and usage of various energy resources. Suggestions are made for incorporation of these methods in site planning for power production facilities and in devising pollution control strategies. Evaluation of general environmental impact of various methods of power production is discussed in the context of decisions made during planning processes. (RE)

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# ENERGY UTILIZATION AND ENVIRONMENTAL HEALTH:

## METHODS FOR PREDICTION AND EVALUATION OF IMPACT ON HUMAN HEALTH

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# PREFACE

The production, distribution and use of energy is a process basic to an industrial society. A variety of socio-economic criteria must be considered when choosing how we are to best utilize our energy resources. Of great significance in such considerations is the prediction and evaluation of existing and potential human health effects due to energy sources and usage. Indeed, adverse health impact exercises veto power over other criteria if construction or operation of energy facilities produces a nonviable environment.

Despite its importance, little attention has been given to health questions in site planning of energy production facilities or pollution control strategies. There have only been limited efforts toward prediction of the environmental health impact of pollutants on populations who may be exposed to such materials. Also of considerable concern is the advent of new processes and technologies for energy production which have their own unique pollution potential.

The purpose of this workbook is to explain and demonstrate evaluation methods which can be used to incorporate public health considerations into the planning process. We believe that these methods will be helpful in the evaluation of environmental impact. We also hope that their publication will stimulate others to develop even more meaningful measures of human health response to environmental stressors.

The development of this workbook has been supported in part by a grant (G007602055) from the Office of Environmental Education, U. S. Office of Education, U. S. Department of Health, Education and Welfare whose support is hereby acknowledged. Mr. Alfred Rosenbloom aided greatly with format, printing and editing and his assistance was much appreciated. Most particularly, great thanks are due to Mrs. Vera Donlan for her organization of the references and typing of the many drafts

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R. A. Wadden
Chicago

March 1977

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# ENERGY AND ENVIRONMENTAL HEALTH

B. W. Carnow

The explosive growth of urbanized industrial societies with attendant use of fossil fuels for the production of electricity, the fabrication of commodities, and the movement of passengers and goods has created serious problems of environment contamination. The challenge we face is to achieve and maintain a healthful environment in the apparent absence of sufficient sources of clean fuel and in the presence of continuing increased demands for heavy industrial and consumer uses of energy.

Some pollution accompanies any energy production and use. Therefore, the selection of energy sources and uses must be considered with care. To assess the options rationally, health cost of a particular energy source must be ascertained and to do this a number of critical questions must be answered. Mainly, how many people are adversely affected by respective energy sources? What is the range of physiological effects? Who in the population are most vulnerable? What might be the additional adverse effects of an increase in pollutants indicated by a proposed change in methods or levels of energy production? If we approach intolerable levels of environmental contamination how do we assess priorities for the generation and uses of energy?

To answer these questions it is first necessary to understand some basic concepts of environmental interactions with human populations. Humans, as biological organisms with varying degrees of resistance and adaptive capacities, are in continuous struggle to control the environment or adapt to it. Any factor in the environment which increases its hostility or anything lowering the resistance of humans decreases adaptability. For example, it is expected that the effect of irritation from toxic gases is strongest on those with inadequate cardiac or pulmonary reserve. Consequently, those with adaptive capacity weakened by serious lung disease, heart disease, asthma or other advanced chronic diseases have been the usual subjects of studies of the effects of air pollutants on humans. Studies carried out by the U.S. Environmental Protection Agency (9-14) and scientific workers in Chicago, Los Angeles, and other cities in the United States (1-4, 6-8), in addition to work in other countries, suggest that frequency of cardiac and respiratory illnesses and death is associated with pollution levels. At every level of pollution, and not at some defined threshold, it appears that, depending upon the adaptive reserve of the individual, someone becomes ill and someone's life is shortened. Although there may be a biological threshold for tissues, the dose-response relationship, even for individuals, will vary with aging, acute illness, stress, fatigue, increased physical activity, changes in nutrition and many other factors. In a large heterogeneous population, there is such marked dose-response variability that no single population threshold can be defined. Thus the important question in relation to morbidity and mortality becomes: How many people are affected at each level of each pollutant?; (or alternatively), What are the numbers at high risks?

In considering host-environment dynamics, the total body burden is a critical factor. Three major areas are involved and all of them must be considered in defining acceptable levels of pollutants.

First, although environmental standards for a single pollutant may be set for a single source adequate to prevent most toxic effects, accumulation of this pollutant from multiple sources may increase the body burden sufficiently to produce negative health effects. For instance, lead enters the body through ingestion and inhalation, utilizing many transporting agents including water, food, paint, street dust, volatilized airborne lead from automobile gasoline, and industrial sources.

Second, in assessing total body burden, the sum total effect of a number of different toxic substances acting on a single organ should be considered. For example, in a nation where 60,000,000 people smoke, we must consider the additive or synergistic effects of this irritant along with the concurrent exposure to $NO_2$, $SO_2$, oxidants, hydrocarbons, and other pollutants, all of which target on the lung. Alcohol, solvents, and lead are also substances that may act synergistically and these target on the liver. We must recognize the possibility that toxic substances may enter into chemical reactions that increase the activity of pollutants. For example, $SO_2$ plus particulate in the presence of high humidity, with manganese acting as a catalyst reacts to form sulfuric acid.

Third, we must consider the impact of other environmental stressors on the total body burden in addition to those imposed by industrial and community toxins. Studies suggest that such stressors as low socioeconomic status with its attendant inadequate medical care, overcrowding, poor housing, poor heating, and poor nutrition enhance the effect of toxic substances by reducing host resistance (5,15).

In the past, many of these health considerations have been overlooked in the planning process. The purpose of this workshop is to present and discuss mothods to incorporate such environmental health considerations into our evaluation of facilities which produce and utilize energy.

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# SOURCES, RECEPTORS, AND THE ENVIRONMENT

R. A. Wadden

The present American lifestyle requires use of considerable amounts of air, water, energy and mineral resources. On a per capita basis, Americans use 30 times as much coal, 40 times as much crude steel, 60 times as much refined copper, and 80 times as much primary aluminum as are used in underdeveloped countries (1). How these resources are maintained or destroyed, dissipated or conserved, depleted or restored have significant implications for human health. How our societal needs can be provided for without harming ourselves is a legitimate requirement of the planning process.

Perception of whether or not a given project is of societal worth depends on comparison of the project's characteristics with one or more criteria which an individual, or groups of individuals, consider to be important. Types of projects that require such evaluation include those to do with transportation (shopping centers, highway networks, airports); and with energy production and use (nuclear and fossil fuel power plants, fusion and solar power, coal and oil conversion processes). Criteria which are often considered in the planning process are utility, accessibility, efficiency, and job and profit potential. Criteria which are rarely or cursorily applied include inconvenience to non-users, degradation of natural resources to non-valuable end products (such as the use of natural gas to produce carbon black), pollution potential, and most significantly, risk to human health.

While all the above factors, as well as others appropriate to a specific project, need to be taken into account, the impact on human health effectively has veto power over all the rest. Establishment of a non-viable environment is self-defeating for any project.

This discussion outlines an organized approach to evaluating the human health implications of projects which have the potential for release of hazardous materials into the environment.

In order to put in perspective the interaction of hazardous discharges into the environment with human health it is helpful to consider the relationships represented in Figs. 1 and 2. Pollutants are discharged from pollutant sources. These discharges are commonly called emissions if they enter the air environment and effluents if entering the water environment. The amounts of emissions and effluents are best described by using a mass/time rate, such as pounds per hour or tons per day. As soon as pollutants are released into the environment they are immediately mixed with whatever materials already exist in the air or water (baseline concentrations). In addition, they undergo chemical and physical changes both because of their interaction with other pollutants, and because of the nature of the receiving air or water. For air, solar radiation, and meteorologic effects, such as temperature inversions and windspeed, will be important. For water, hydrologic considerations will affect the dissipation or concentration of a pollutant.

Pollutants in air (or water) environments are characterized by measuring their mass per unit volume of air (or water), i.e., their concentration. This concentration is related to a particular averaging time, which corresponds to the time interval of sample collection, or the time over which a number of samples are combined. The relationship between source emissions or effluents and environmental concentrations can be estimated by using air quality or water quality models.

The most important consideration of air and water pollution is the consequences that their environmental concentrations may have for human receptors. The relevant steps in evaluating such consequences

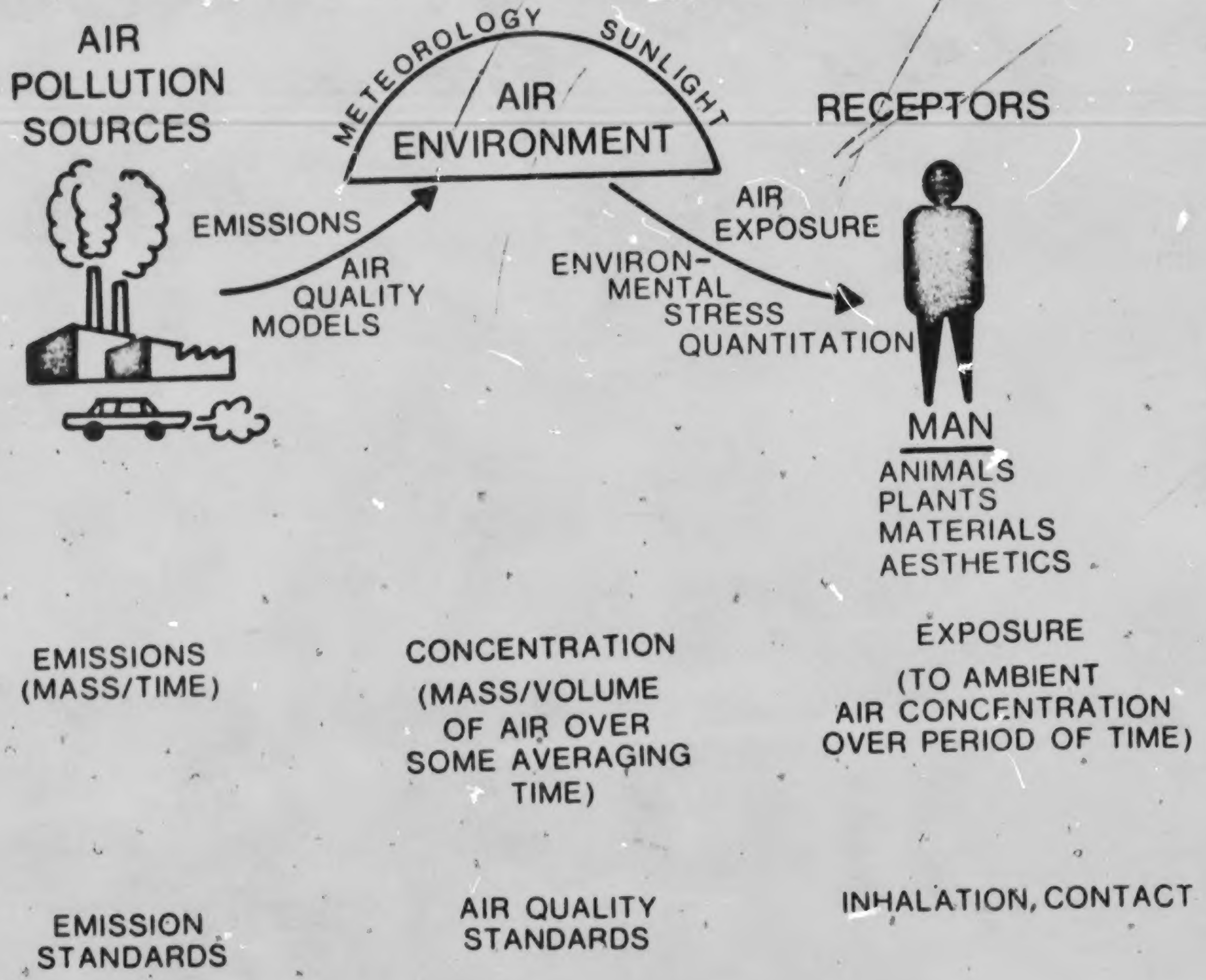


Figure 1. Sources, receptors

and the air environment.

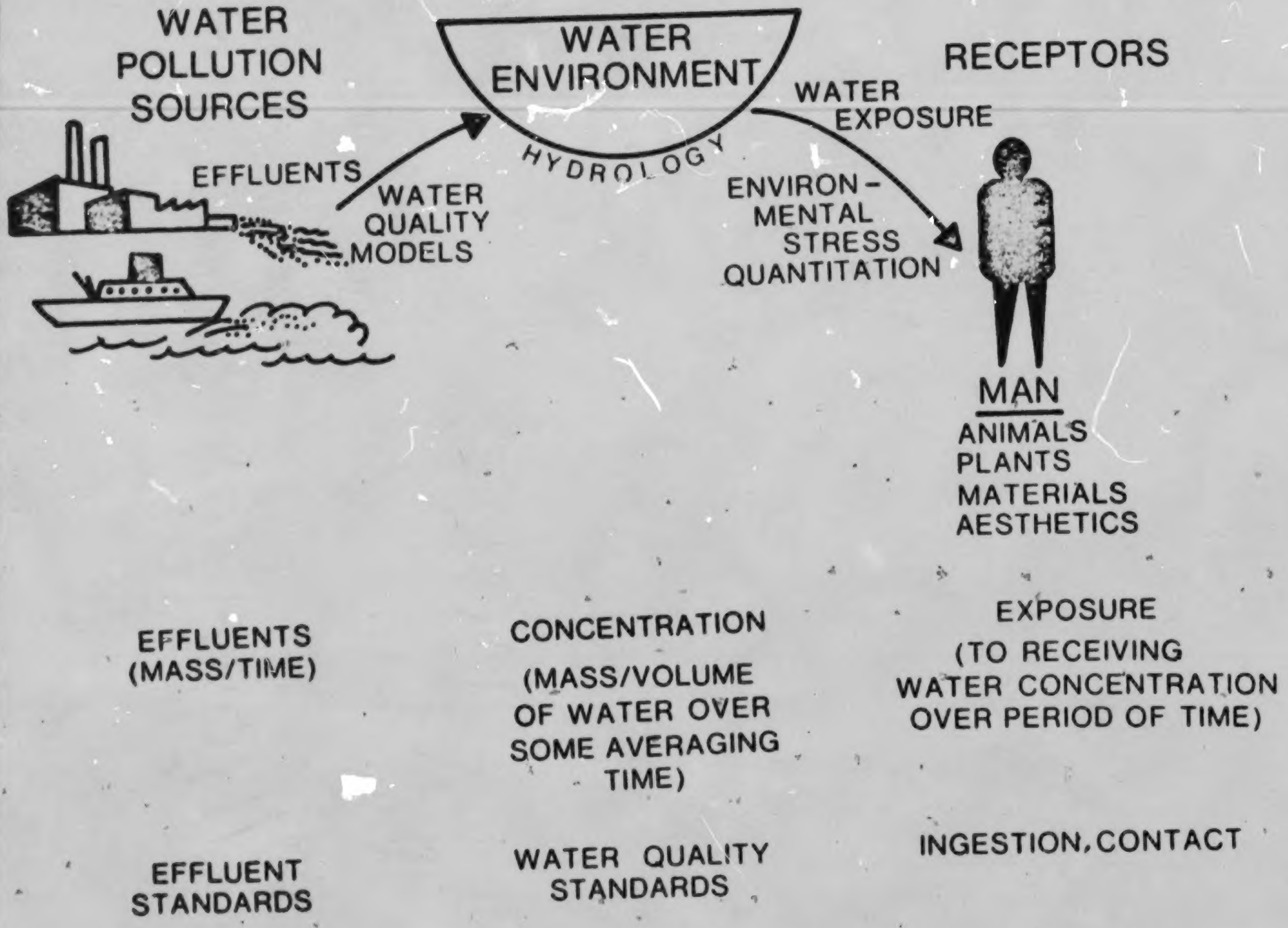


Figure 2. Sources, receptors

and the water environment.

require asking the following questions: What individuals are most at risk to the expected environmental hazards?; how many of them are there?; where are they located with respect to the pollution source?; and how much are they likely to be affected by the additional environmental stress?

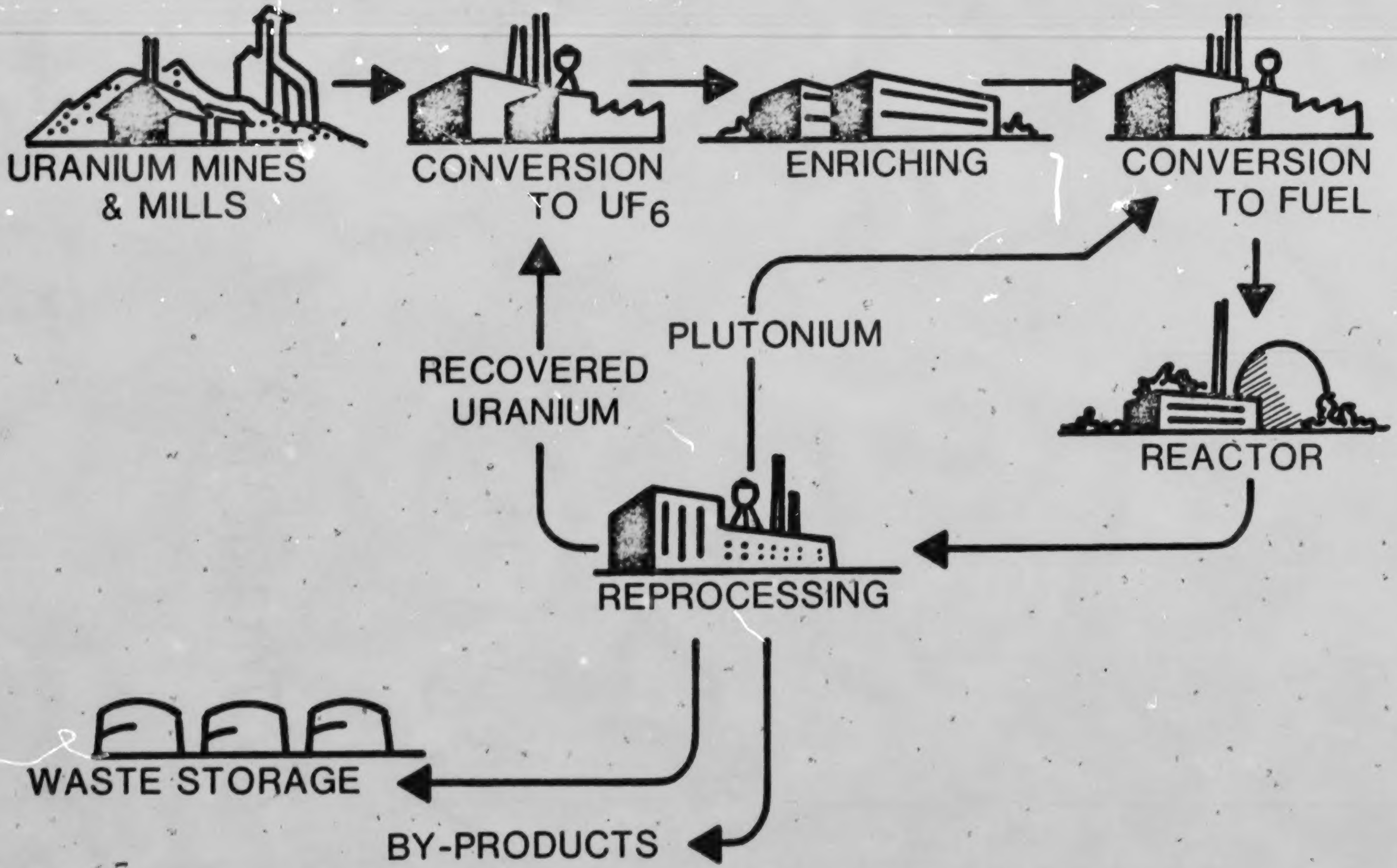
The consideration of all the factors noted above should be incorporated into the environmental impact analysis of a new energy source or use. Such an analysis may cover only a specific source and site evaluation with limited geographical considerations, or it may involve an evaluation of a total energy cycle. Figure 3 indicates such a cycle for nuclear fuel. At each step there are pollution and health consequences to be considered. Our focus during this workshop will be mainly on health implications of specific sources or planning alternatives. However, the same procedures can also be applied to the steps of an energy cycle.

To briefly summarize then, the major steps in the overall review process (2) are:

(a) Identification of hazards - raw material, type of process, method of operation,
(b) Quantitation of hazardous wastes and byproducts - air water, soil,
(c) Measurement of "baseline" conditions of the environment - present air, water and soil quality,
(d) Dispersion of hazardous discharges - what and how much goes where,
(e) Identification, enumeration and location of populations at risk,
(f) Estimation of exposure levels and related health effects.

Figure 3.

# THE NUCLEAR FUEL CYCLE



Each of these categories will be discussed more completely in later chapters. It is important that the items listed above become a regular consideration of those planning and deciding upon projects with pollution potential. In addition, such considerations should be more specifically incorporated into legislation and regulations involving pollution sources. Finally, public agencies should be encouraged to investigate and follow-up the health implications of existing and new projects which discharge environmental hazards. Our concern for material prosperity should not cause us to neglect our responsibilities for human health.

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# RECEPTOR CHARACTERIZATION:

# IDENTIFICATION OF HYPERSUSCEPTIBLE POPULATIONS

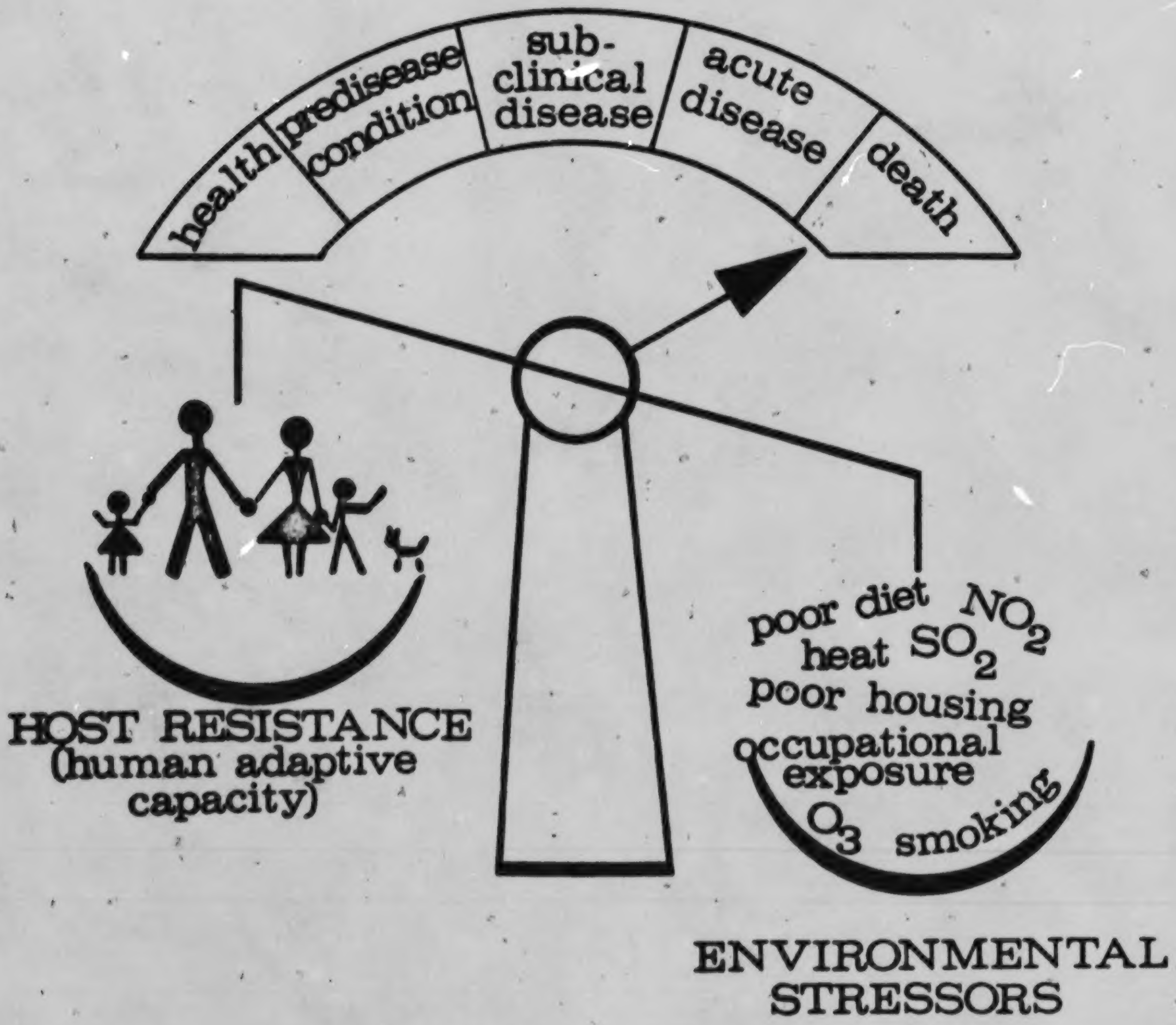
B. W. Carnow

The state of public health is not static. It fluctuates in response to environmental stress according to individual sensitivities and the range and strength of environmental forces acting singly or in combination, successively or at the same time. A simplified representation of the effect on the health continuum of a disturbance in the balance between human adaptive capacity and environmental stressors appears in Figure 1. The relationships of energy production and use to public health are discussed in the framework of this concept. Our task is to define the nature and magnitude of environmental stressors associated with energy applications and to determine their effect on individuals with varying adaptive capacities.

Tables 1 and 2 list many of the factors which have been found or might be expected to increase susceptibility at levels which apparently do not affect healthy individuals. Several factors affect the sensitivity of a population. One set of risks inherent in the individual include age, sex, general state of health, genetic predisposition and pre-existing disease. Another set relates to activity or place. Certain occupations present characteristic hazards. Other risks are characteristic or typical habitats. Locations and activities, including personal habits such as cigarette smoking or eating patterns, frequently reflect an economic status with a high risk of disease.

Those hypersusceptible to stress are a large segment of the U.S. population. Specifically they include: 1) Those inhabitants of congested urban area typified by psychosocial tension, poor nutrition, overcrowding, poor heating (for example, the use of heaters themselves frequently produce carbon monoxide and nitrogen compounds) and other

Figure 1. Environmental Stressors in the Public Health Continuum



## Table 1. Populations at High Risk to Environmental Pollutants Because of Cardiac Deficiencies

**Genetically Deficient**

- *Hypercholesterolemia
- *Diabetes with atherosclerosis

**Congenital Disease**

- *Tetrology of Fallot
- *Atrial or ventricular wall defects
- *Valvular defects

**Chronic Disease**

- *Cor Pulmonale (Secondary to chronic obstructive pulmonary disease)
- Hypertension with left ventricular disease
- Coronary insufficiency (*with or without angina)
- Rheumatic heart disease
- *Congestive heart failure (Secondary to atherosclerotic heart disease)

**Other Environmental Factors**

- Cigarette smoking
- *Heavy work exposure

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*No data on $SO_2$ effects on these populations - but might be expected to be at high risk.

## Table 2. Populations at High Risk to Environmental Pollutants Because of Pulmonary Deficiencies

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### Genetically Deficient

- Asthma
- Cystic Fibrosis
- *α, anti trypsin deficiency
- Cystic disease of lung

### Chronic Disease

- Chronic bronchitis
- Emphysema
- *Kyphoscoliosis
- *Advanced (active or inactive) tuberculosis, histoplasmosis
- *Bronchiectasis

### Other Environmental Factors

- Cigarette smokers
- Workers exposed to dusts, fumes, smokes, aerosols, gases

### Others

- *Prematures
- *Newborns
- Preschool children
- The poor, and those exposed to other environmental stresses such as overcrowding, malnutrition, poor heating
- *Obesity

---

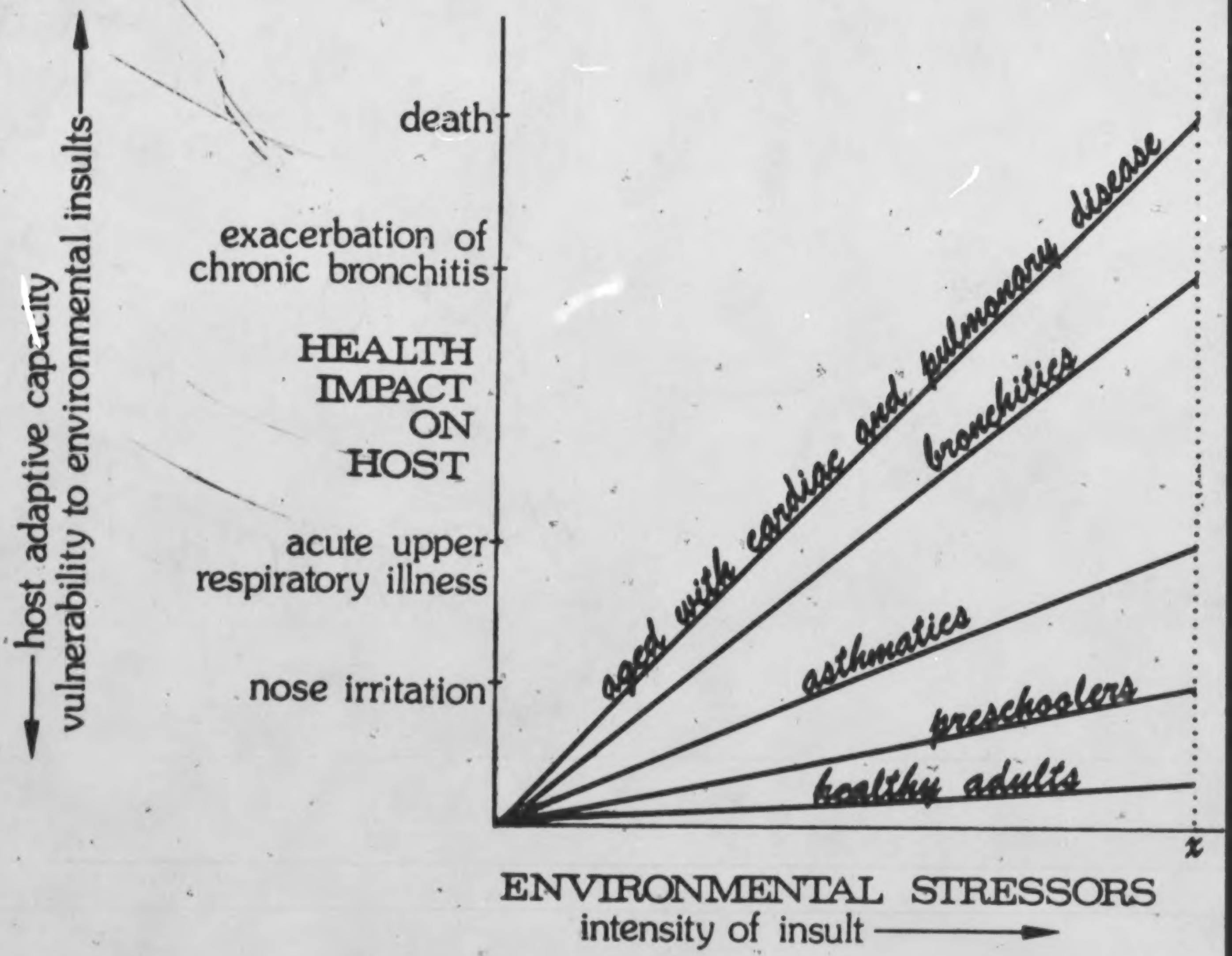
*No data - but might be expected to be at high risk.

factors associated with poverty. 2) Fetuses, neonates and pre-school children. 3) The geriatric population whose medical history includes a high frequency of cardiovascular and cardiopulmonary disease. 4) Those suffering from advanced bronchitis, emphysema, asthma, or other diseases whose symptoms worsen upon exposure to polluted air. 5) Cigarette smokers and those exposed to large amounts of cigarette smoke, especially those already sustaining a body burden of a variety of noxious gases and particulate matter. 6) Those involved in very heavy physical exertion requiring optimal energy transport. 7) People exposed by occupation or habitat to hazardous stresses, such as asbestos, beryllium or cotton dust, which may lead to diffuse scarring, fibrosis and reduced elasticity of the lungs. All of these conditions reduce the capacity to deal with additional environmental stress. 8) Those with genetic handicaps such as asthma, alpha-1-antitrypsin deficiency or cystic fibrosis.

Figure 2 is an idealized representation of population responses to environmental stressors. As the degree of stress increases, i.e., the burden of pollution, the severity of the effects (or the population affected) increases. A given level of stress will have different effects on given sub-populations. For example, pollution level x in the figure may cause the death of those most at risk, the aged with advanced cardiac and pulmonary diseases; it might exacerbate chronic bronchitis; cause upper respiratory infections; increase the numbers and frequency of asthmatic attacks; or increase nose or sinus irritation in pre-school children. Healthy adults would not be affected perceptibly by level x. One must note in regard to this, however, that the effects of long time exposure to low levels even on the healthy have not been adequately documented in adults. A long term British study of a large number of people, who as children were raised in high pollution areas, suggests that exposure early in life may lead to respiratory difficulty as an adult. (1)

Figure 2 is, or course, merely schematic. Conceptually it applies to any specific environmental stressor. For air pollutants such as sulfur

Figure 2. Population Response to Environmental Stressors



dioxide the population at highest risk would be the aged with cardiac and respiratory disease. For water viruses those at high risk who should be studied might include very young children and those with liver disease who might be highly susceptible to hepatitis virus. Clues to who should be considered may be derived from examining the organ or organs most likely to be stressed by the pollutants, and then defining in the general populations those groups with poor adaptive capacity of this organ. If we consider the liver in this regard, hypersusceptible groups include those with alcoholism, individuals with fatty livers due to malnutrition, and those whose livers have undergone inflammatory changes due to heavy exposure to carbon tetrachloride or other similar solvents.

The diagram does not weight the subpopulations according to their distribution in the general population. Also it does not demonstrate that, while x shortens the lives of the highest risk populations, it may also decrease the resistance of others, e.g., pre-school children, to the end that some will be high risk in later years with their lives possibly shortened.

The concept we have adopted in using this model of dose-response is that in general the response to a stress in a population is not pure chance but is determined by definable factors. The part of the population which responds to low doses of a given stress is a specific, pre-determined and possibly identifiable category as previously described. As the stress increases a response may be involved. Even though the population dose-response curve cannot be described exactly, the theory that it is composed not of a single response but of a series of responses by distinct subpopulations can be quite useful in designing health programs. Such programs must consider not only the effects of environmental stress on health but also who is affected. Society appraises the same effect differently as applied to a child, a young adult or a retired person.

The physical and biological sciences are able to contribute to estimates of the bioenvironment's basic effects on public health. The social sciences assist in assigning values to specific effects on subpopulations. Society as a whole, however, decides how to use such information. It must again be emphasized that the concept of a threshold effect exemplified by defining "safe levels" to a heterogeneous population has made it impossible over the years to quantitate populations at risk. It implies a "safe for everyone" level below this number and an "unsafe for anyone" level above this number. Rejection of this concept makes it possible to begin to define variations in risk at every level of pollution. There may be a "safe" level in a sense that effect may never be measurable. At virtually every level of stress, however, some die, some may fall ill, and some suffer minor insults depending on individual capacity to adapt.

Figure 2 points up the need for recognizing health and disease as a continuum and not as sharply defined entities. WHO's definition of health (2), ". . . not only the absence of disease but a state of physical and mental well being . . ," is hardly achievable in an urban center with all of its physical irritants, emotional stress, noise and other similar stressors. The long incubation periods for most environmentally-related diseases, including chronic pulmonary disease and cancer, points up the need to focus on preclinical factors and subclinical disease.

Certain environmental effects have yet to be shown to lead to chronic or progressive disease, although their effects on individuals warrant attentive monitoring. Examples of these include elevated carboxyhemoglobin levels in smokers and urban dwellers, decreased respiratory function in children in highly polluted areas, and lower antibody titers and influence of viruses among smokers compared to nonsmokers. Procedures which measure change in the small peripheral airways are how being used to evaluate pollutant effects on lungs. These procedures indicate that small airways are affected early and at levels of pollution much lower than heretofore thought. Previous evaluation methods considered only large airways and concluded that no health effect occurred at these levels.

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# RECEPTOR CHARACTERIZATION:
# ENUMERATION AND LOCATION OF HYPERSUSCEPTIBLE POPULATIONS

Donna O. Farley

In order to estimate health impacts of environmental pollutant sources, the population groups most susceptible to the pollutants emitted need to be identified, enumerated and located relative to the sources. Their predicted health effects would be estimated by the following: (1) the numbers of high risk persons exposed, (2) the levels of pollutant exposure received and (3) a measure of physiological response to that exposure.

Within any given area, population densities will not be uniform. The more precisely the distributions of each hypersusceptible population can be quantified, the more accurately the health impacts of pollutants within an area can be predicted. A frequent procedure used is to divide an area of interest into subareas, thereby providing a geographical grid within which high risk populations can be enumerated.

Groups which are selected for enumeration must be quantifiable for these areas and subareas, so the availability of data at the relevant geographical scale must be assessed. Also, the applicability of enumeration techniques to that scale must be determined.

Techniques for enumerating hypersusceptible populations fall into the three categories presented below. One or more of these techniques may be used, as necessary, to estimate the sizes of the various populations included in a health impact analysis.

## DEMOGRAPHIC CHARACTERISTICS

This technique enumerates persons whose hypersusceptibility can be defined by demographic characteristics, such as youth or old age, with the primary sources of data being the U. S. Bureau of the Census (8) or state census data. Both the U. S. Decennial Censuses and national population estimates for intermediate years provide data on the total population for limited geographical areas. Characteristics of the population, such as age, race or income, are also available in printed census reports or on computer tapes.

The census data are available on very small geographical scale: by blocks, census tracts or enumeration districts. These units, however, have geographical boundaries unique to the Census. Either their boundaries must be used for subareas, or census data must be translated to the grid system being used. Extensive translation will require a computer and probably access to census data stored on tape rather than in published reports.

## FACILITIES OCCUPIED BY HYPERSUSCEPTIBLE POPULATIONS

Among the facilities included in this category are acute care hospitals, long-term care facilities, day care centers and schools. Because data must be expressed as the number of hypersusceptible persons in each subarea, a detailed listing must be made of all facilities, including their exact locations and capacities for occupancy. The capacity (or actual average occupancy, if available) of each facility is then assigned to the subarea in which the facility is located. (See references 2 and 9 for examples of use of this method.)

Because of their very specific site locations, high risk facilities can be used to enumerate hypersusceptible populations on virtually any geographic scale. Unlike census data, however, these data do not necessarily represent persons living in those locations. For example, people may occupy schools from six to eight hours per day over a period of years, but for hospitals the occupancy may be for twenty-four hours for from one day to several months. These differences result in different pollutant exposures and effects.

## PREVALENCE OF HEALTH CONDITIONS

People with health conditions which render them hypersusceptible to pollutant exposure are the most important high risk groups, particularly when acute health impacts are being estimated. These are the first people affected by elevated pollutant concentrations, and they may be expected to have more obvious dose-response patterns than hypersusceptible populations estimated by facility or age.

Unfortunately, direct estimates of prevalence (number of people with a given health condition) for local areas are not available, due to the high costs for surveys to develop them. Therefore, other methods must be used to obtain local prevalence estimates for the health conditions being measured.

At the national level, prevalence estimates for various health conditions are available from the National Center for Health Statistics (NCHS) and are published in its "Vital and Health Statistics" series of reports (4). By applying the appropriate U. S. prevalence rate estimates to local population data the number of local people in an area with the conditions being measured can be estimated. It should be remembered, however, that the accuracy of the estimates will be determined by how closely the characteristics of the local population match the national sample.

The most simple application of this approach is to use the national crude prevalence rate of a condition as the local rate. This method has been the most commonly used one and, lacking other techniques, is probably the best available. It does not, however, take into account local differences in population characteristics (e.g., age or race) which have a significant influence on a condition's prevalence.

A technique called synthetic estimation has been developed and tested by the NCHS (6). This technique accounts for variations in local population characteristics when making local prevalence estimates by dividing the population data into subgroups by characteristic (e.g., age, race and sex) and applying specific prevalence rates (usually national level) to each of the subgroups. The subgroups are called demographic cells (α cells). The sum of the populations in the cells is equal to the total local population, and the total local prevalence of a health condition is the sum of the estimated prevalence in all of the demographic cells.

Using this technique two models for estimating the local prevalence of chronic bronchitis, emphysema and asthma have been developed and tested by the author (3). These diseases were selected because they are prevalent diseases in the U. S. today. Not only do chronic bronchitis, emphysema and asthma have major impacts in disability and death, but also patients with one or more of these diseases are hypersusceptible to air pollutants affecting lung function (1).

The first model is simply a synthetic estimation. It uses local population data divided into cells by age, race and sex and can therefore be used for any local area for which population data are available in that form. It is expressed as:

$$\tilde{x}_{Ld} = \frac{10^5}{P_L} \sum_{\alpha=1}^{k} (p_{\alpha L} \bar{x}_{\alpha d}) \qquad (1)$$

where: $\tilde{x}_{Ld}$ = the local synthetic estimate of prevalence rate per 100,000 population for disease d; $\bar{x}_{\alpha d}$ = the U. S. estimate of the proportion of the population in the αth cell; $P_L$ = the total local population; and d = the disease being estimated (chronic bronchitis, emphysema, or asthma); and $P_{\alpha L}$ is the local population in the $\alpha$th cell.

The NCHS has developed U.S. prevalence rate estimates $(\bar{x}_{\alpha d})$ for 1970 each disease by age, race and sex (5). They are given in Table 1.

The second model is designed to convert local death rate data into prevalence rate estimates. This is done by calculating estimates of local ratios of case fatality (i.e., ratios of the annual deaths from a disease to the number of cases of the disease) and applying those ratios to local actual death rate data. The model is given as:

$$\tilde{X}'_{Ld} = \frac{\tilde{X}_{Ld}}{\tilde{Y}_{Ld}} (\bar{Y}_{Ld}) \qquad (2)$$

where: $\tilde{X}'_{Ld}$ = the death rate conversion estimate of local prevalence rate per 100,000 population for disease d; $\bar{Y}_{Ld}$ = the actual local death rate per 100,000 population for disease d; $\tilde{Y}_{Ld}$ = local synthetic estimate of death rate per 100,000 population for disease d. Both $\tilde{X}_{Ld}$ and $\tilde{Y}_{Ld}$ are synthetic estimates. Their ratio is a population-weighted ratio of cases to deaths, which converts an actual local disease death rate, $\bar{Y}_{Ld}$ into a prevalence rate.

The synthetic death rate estimate, $\tilde{Y}_{Ld}$, is of the same form as equation (1). It is obtained by applying a cell-specific rate, $\bar{y}_{\alpha d}$, to the local population in each cell, where $\bar{y}_{\alpha d}$ = the U.S. estimate of the proportion of the population in the $\alpha$th cell dying annually from disease d. The U.S. death rates by age, race and sex $(\bar{y}_{\alpha d})$ for 1970, obtained from the Census and Vital Statistics of the United States, are given in Table 2 (7,8). The $\alpha$ cells for $\bar{y}_{\alpha d}$ are identical to those for $\bar{x}_{\alpha d}$ given in Table 1, so the two sets may be used together in the model.

Table 1. Estimated Proportion of U. S. Population with Chronic Bronchitis, Emphysema, and Asthma, by Age, Race and Sex, 1970 ($\bar{x}_{\alpha d}$)

| Chronic bronchitis | Under 17 | | 17-44 | 45-64 | 65-over |
|---|---|---|---|---|---|
| W. male | .0469 | | .0175 | .0295 | .0493 |
| W. female | .0365 | | .0308 | .0431 | .0375 |
| All other | .0227 | | .0142 | .0235 | .0260[a] |
| **Emphysema** | | | **Under 45** | | |
| W. male | | | .0013 | .0238 | .0639 |
| W. female | | | .0009 | .0068 | .0124 |
| All other | | | .0001[a] | .0043[a] | .0026[a] |
| **Asthma** | **Under 6** | **6-16** | **17-44** | | |
| W. male | .0343 | .0373 | .0252 | .0281 | .0415 |
| W. female | .0211 | .0238 | .0274 | .0355 | .0306 |
| All other | .0364 | .0392 | .0252 | .0445 | .0429 |

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[a] Figure does not meet NCHS standards of reliability or precision (>30% relative standard error).

Table 2. U. S. Death Rates per 100,000 for Chronic Bronchitis, Emphysema, and Asthma, by Age, Race and Sex, 1970 ($\bar{y}_{\alpha d} \cdot 10^5$)

| Chronic bronchitis | Under 17 | | 17-44 | 45-64 | 65-over |
|---|---|---|---|---|---|
| W. male | .3509 | | .2398 | 5.6570 | 36.5959 |
| W. female | .2456 | | .1733 | 2.2030 | 8.4326 |
| All other | .6472 | | .3357 | 3.5404 | 8.4718 |
| **Emphysema** | | | **Under 45** | | |
| W. male | | | .2802 | 26.7338 | 168.9975 |
| W. female | | | .1762 | 6.3184 | 21.4513 |
| All other | | | .4392 | 10.5008 | 36.7689 |
| **Asthma** | **Under 6** | **6-16** | **17-44** | | |
| W. male | .2441 | .1488 | .2705 | 1.5071 | 4.2246 |
| W. female | .1624 | .1662 | .5469 | 1.9837 | 3.6126 |
| All other | .7799 | .5812 | 2.0878 | 5.3949 | 5.5326 |

The second model was developed when it was ascertained that synthetic estimates of prevalence and mortality using age, race and sex as determinants do not fully explain variations in actual local prevalence and mortality for these diseases. This model is expected to yield more accurate estimates than the first model for two reasons: (1) it develops estimates from actual local mortality data; and (2) it takes into account local population characteristics when converting actual local death rates into predicted prevalence rates.

In the model, local actual death rates averaged over a period of several years should be used to minimize effects on the estimates of random fluctuation over time in the local mortality data. If reliable mortality data are not available, the synthetic estimation model may be used.

Although they yield biased estimates, the two prevalence rate estimation models have predictive capabilities superior to that of crude rates applied to local populations, and are useable with existing census, morbidity and mortality data.

Evaluation during model development indicates that the bias of the estimates is not affected by the size of the local population. They may, therefore, be used for small areas or subareas to enumerate populations who are hypersusceptible to pollutant stress due to chronic bronchitis, emphysema or asthma. The bias in the estimates is probably due to omission from the model of other disease determinants, for example occupational exposures or smoking. This is true for both prevalence and death rate estimates and the ratios of case fatality calculated from them.

What will probably determine the applicability of these models to any given location will be the availability of the necessary local population and mortality data. Local population data by age, race and sex is essential to the models, and the mortality data will enable better estimates to be made through use of the death rate conversion model. If neither model can be used, then crude U.S. rates are useful. Depending on the local population data, prevalence rates which are only specified by age or race may also be used. They would generate estimates which would be expected to be somewhat more accurate than the crude estimates.

These synthetic estimation models were developed and tested only for chronic bronchitis, emphysema and asthma. The same models can, however, be adapted to other health conditions for which U.S. prevalence rate estimates are available for specific demographic cells. The cell divisions would be determined by those characteristics which have the greatest influence on a particular condition. Before using new models for other conditions, they should be tested using evaluative procedures similar to those used to evaluate these models (3). It should be determined if the models predict better than crude rates, how biased they are, and if their bias would be affected by the size or characteristics of the populations in the areas for which the prevalence of the condition will be estimated.

## SAMPLE APPLICATION OF PREVALENCE ESTIMATION TECHNIQUES

The synthetic estimation models are demonstrated here by using both of them to estimate the prevalence rates and prevalence (number of people) for chronic bronchitis in ten "counties" in a hypothetical geographical region. A third set of disease prevalence estimates was also calculated using the 1970 crude U.S. bronchitis prevalence rate of 3270 per 100,000 as the rate for each of the ten "counties."

The first step in the estimation procedure is to calculate the synthetic estimates of prevalence rates and death rates. This is done by multiplying the cell-specific rate for each cell by the population in that cell and summing the results for all cells in the total population. The rates used are the proportions of prevalence

($\bar{x}_{\alpha d}$) and proportion of deaths ($\bar{y}_{\alpha d}$) for chronic bronchitis, given in Tables 1 and 2, respectively. An example of this procedure for county 1 is demonstrated in Table 3, which gives the cell-specific populations and the prevalence and death rates resulting from the calculations.

The development of the synthetic estimate and death rate conversion estimate for the same county 1 are shown in Table 4. The synthetic estimates are the prevalence and prevalence rate calculated and presented in Table 3. For the death rate conversion estimate, the prevalence and death rate estimates are used to calculate the ratio of cases to deaths, which is then multiplied by the actual local bronchitis death rate (in this case, a three-year average rate) to yield an estimated prevalence rate.

Results of these calculations performed for each of the ten hypothetical counties are presented in Table 5, along with the estimated prevalence calculated using the crude U.S. bronchitis prevalence rate. Different patterns of disease prevalence (both rates and numbers of people) are obtained from each method, as shown graphically in Figures 1 through 4.

Figure 1 shows the distribution of counties for death rate conversion estimates of prevalence rates. In contrast to this rather wide distribution, all ten counties have estimated rates for both the synthetic estimates and the crude rate estimate (which is 3270 per 100,000) that fall in the 3001-4000 per 100,000 class.

A wider distribution for prevalence values is observed, reflecting the wide range of populations in the ten counties (76,850 to 491,882 total population) from which they are calculated. The three distributions of synthetic estimates, death rate conversion estimates and crude estimates of prevalence are highlighted in Figures 2 through 4. These figures and Table 5 show that the death rate conversion estimates are the most conservative estimates and that the synthetic estimation

Table 3. Population and Synthetic Prevalence and Death Rate Estimates for Chronic Bronchitis by Age, Race, and Sex for County 1

| | Under 17 | 17-44 | 45-64 | Over 65 |
|---|---|---|---|---|
| **Population** | | | | |
| W. male | 14341 | 11956 | 8614 | 2066 |
| W. female | 13783 | 13356 | 8437 | 2183 |
| All other | 295 | 386 | 343 | 90 |
| Total = 76850 | | | | |
| **Prevalence** (Number of persons) | | | | |
| W. male | 674 | 209 | 254 | 102 |
| W. female | 503 | 411 | 364 | 119 |
| All other | 7 | 5 | 8 | 2 |
| Total = 2657 persons | | | | |
| Prevalence Rate ($\tilde{X}_{L1}$) = 3458 per 100,000 | | | | |
| **Death Rate** (Per 100,000 total population) | | | | |
| W. male | .0655 | .0373 | .6341 | .9838 |
| W. female | .0440 | .0301 | .2419 | .3493 |
| All other | .0025 | .0017 | .0158 | .0099 |
| Total ($\tilde{Y}_{L1}$) = 2.416 per 100,000 | | | | |

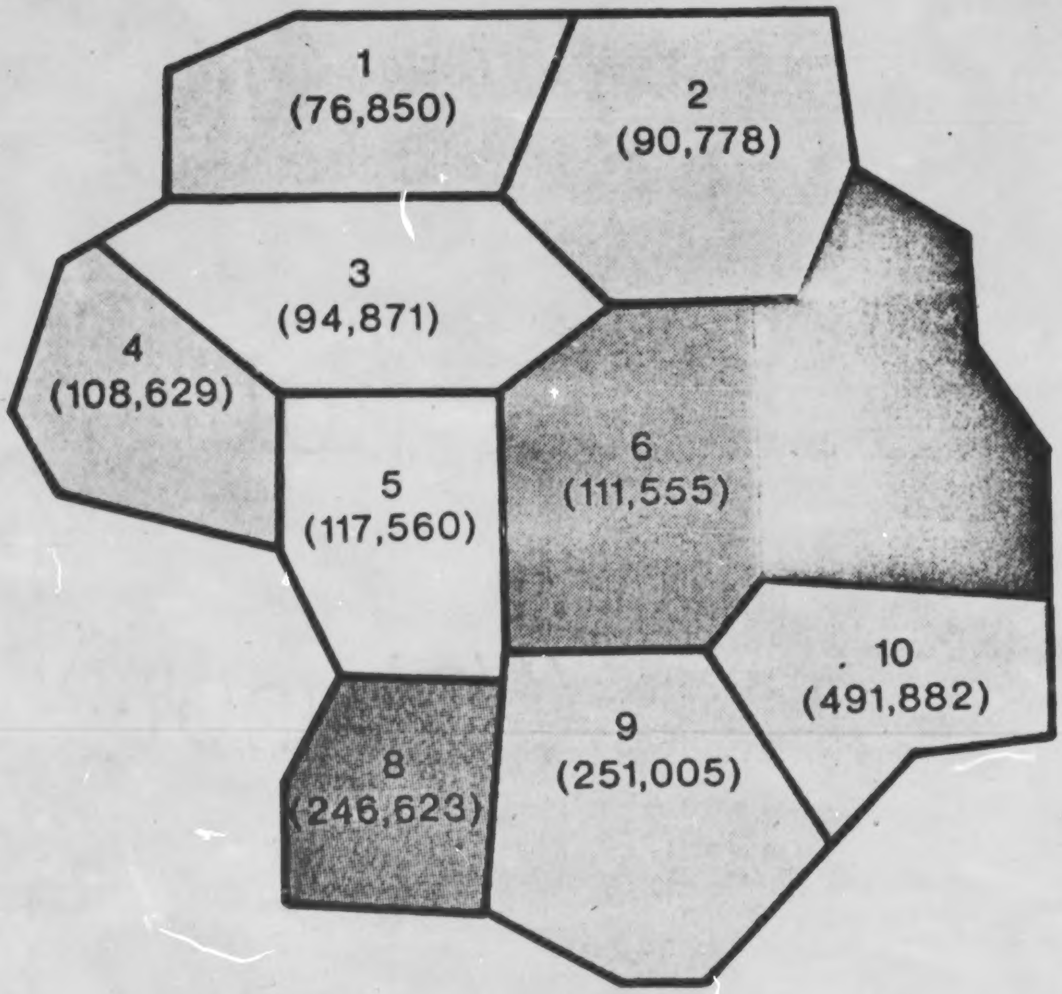
Table 4. Synthetic and Death Rate Conversion Estimates of Chronic Bronchitis Prevalence in County 1

| | Intermediate results | Prevalence rate per 100,000 | Prevalence (No. of people) |
|---|---|---|---|
| 1. Synthetic estimation | | 3458 | 2657 |
| 2. Death rate conversion | | | |
| $\tilde{X}_{L1}$ (per 100,000) | 3458 | | |
| $\tilde{Y}_{L1}$ (per 100,000) | 2.416 | | |
| Ratio-cases to deaths | 1431 | | |
| Actual bronchitis Death rate per 100,000 ($\bar{Y}_{L1}$) | 1.910 | | |
| Prevalence estimates | | 2734 | 2101 |

Table 5. Prevalence and Prevalence Rate Estimates for Chronic Bronchitis by Three Estimation Methods

| County | Synthetic Estimates | | Death Rate Conversion | | Crude Estimate |
|---|---|---|---|---|---|
| | Prevalence Rate $\tilde{X}_{Li}$ | Prevalence (Number of People) | Prevalence Rate $\tilde{X}_{Li}$ | Prevalence (Number of People) | Prevalence (Number of People) |
| 1 | 3458 | 2657 | 2743 | 2101 | 2513 |
| 2 | 3443 | 3125 | 2233 | 2027 | 2968 |
| 3 | 3215 | 3050 | 1919 | 1821 | 3102 |
| 4 | 3408 | 3702 | 2291 | 2489 | 3552 |
| 5 | 3450 | 4125 | 1881 | 2249 | 3910 |
| 6 | 3469 | 3870 | 3968 | 4427 | 3648 |
| 7 | 3411 | 5503 | 4516 | 7286 | 5276 |
| 8 | 3334 | 8222 | 3475 | 8570 | 8065 |
| 9 | 3376 | 8474 | 2929 | 7352 | 8208 |
| 10 | 3402 | 16734 | 2668 | 13123 | 16085 |
| Total prevalence | | 59,462 | | 51,445 | 57,327 |
| Average prevalence rate (per 100,000) | | 3,391 | | 2,935 | 3,270 |

# CHRONIC BRONCHITIS PREVALENCE ESTIMATION FOR TEN COUNTIES IN A HYPOTHETICAL GEOGRAPHICAL REGION



☐ 1001-2000 PER 1000,000

☐ 2001-3000 PER 100,000

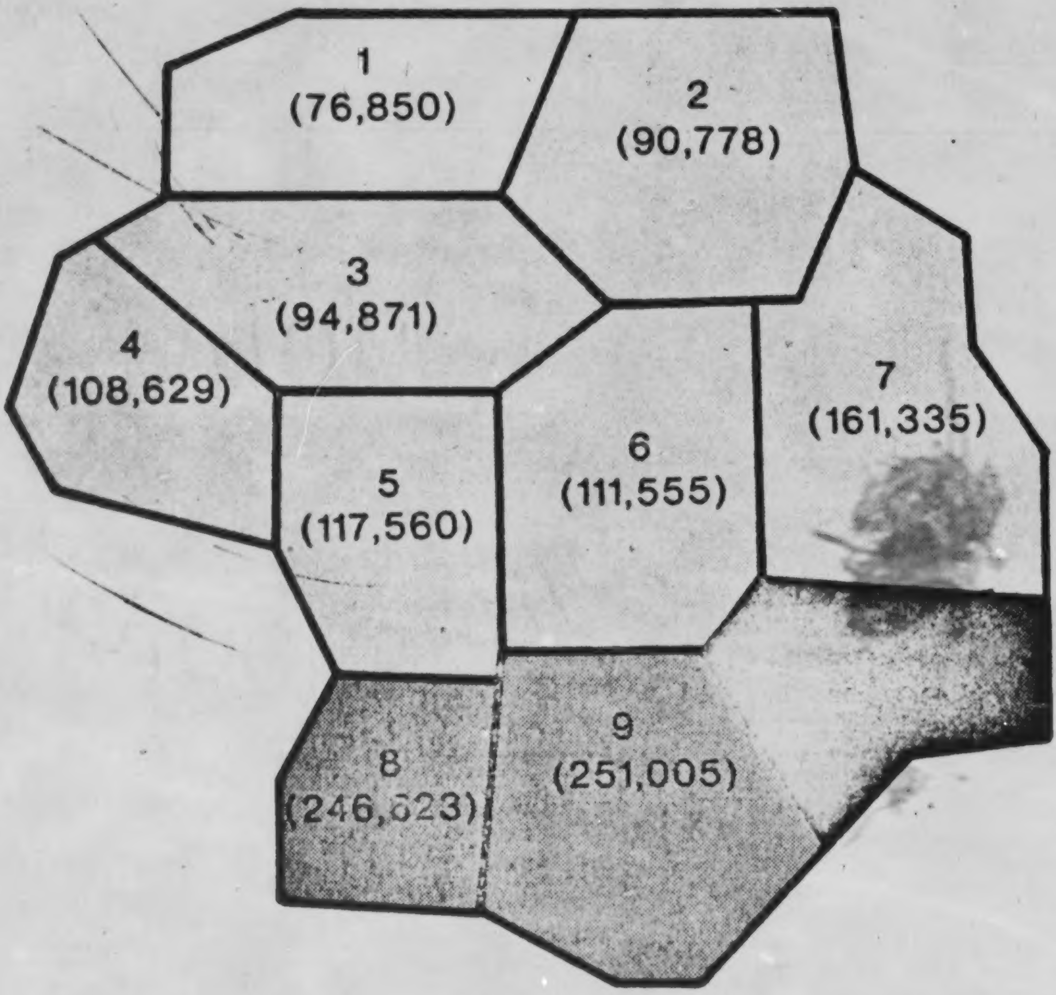
▨ 3001-4000 PER 100,000

■ 4001-5000 PER 100,000

FIGURE 1. DISTRIBUTION OF COUNTY PREVALENCE RATE ESTIMATES CALCULATED BY USE OF THE DEATH RATE CONVEVERSION METHOD

*NUMBER IN ( ) INDICATES THE POPULATION FOR THE COUNTY. THE TOTAL REGION POPULATION IS 1,753,088 PERSONS.

# CHRONIC BRONCHITIS PREVALENCE ESTIMATION FOR TEN COUNTIES IN A HYPOTHETICAL GEOGRAPHICAL REGION



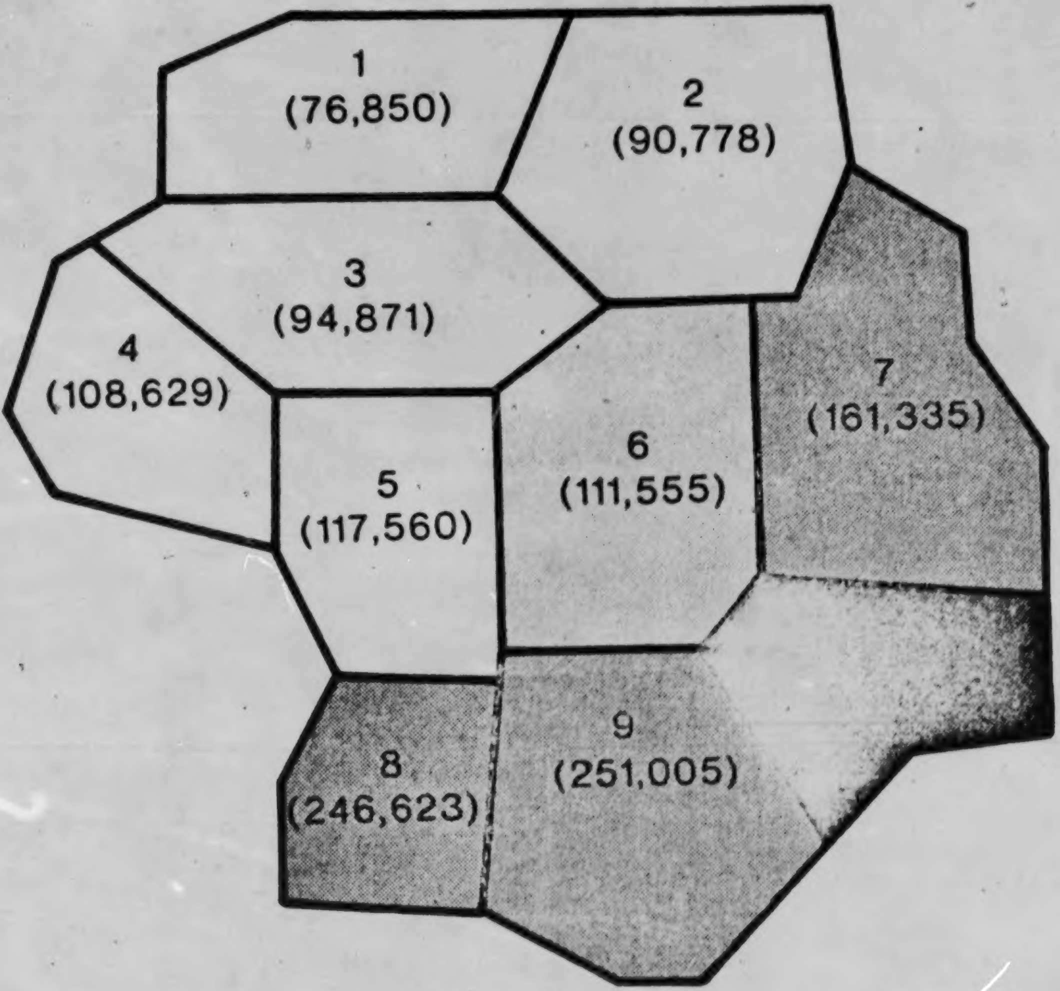
☐ 0-3000 PERSONS ▨ 6001-9000 PERSONS

☐ 3001-6000 PERSONS ■ 9001 PERSONS AND OVER

FIGURE 2. DISTRIBUTION OF COUNTY PREVALENCE ESTIMATES CALCULATED BY USE OF THE SYNTHETIC ESTIMATION METHOD

*NUMBER IN ( ) INDICATES THE POPULATION FOR THE COUNTY. THE TOTAL REGION POPULATION IS 1,753,088 PERSONS.

# CHRONIC BRONCHITIS PREVALENCE ESTIMATION FOR TEN COUNTIES IN A HYPOTHETICAL GEOGRAPHICAL REGION



☐ 0-3000 PERSONS

☐ 3001-6000 PERSONS

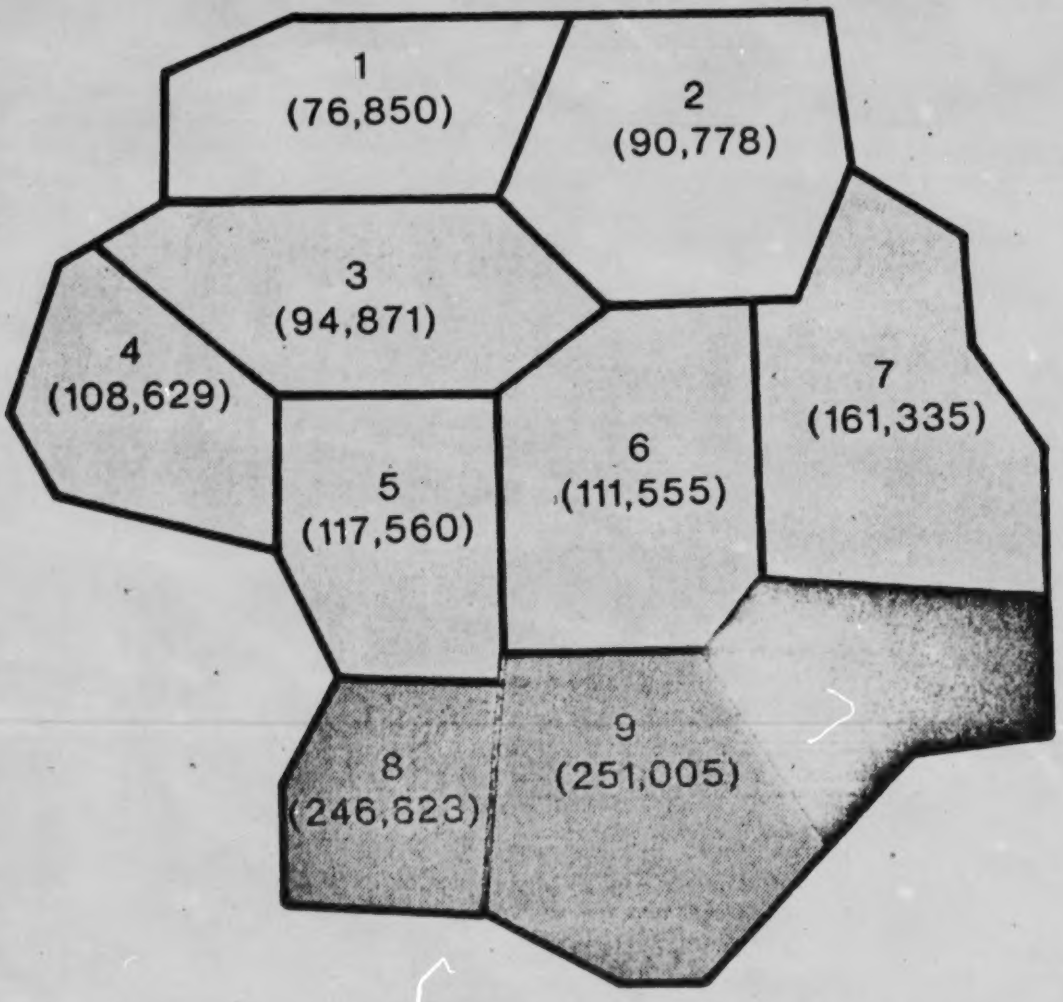
▨ 6001-9000 PERSONS

■ 9001 PERSONS AND OVER

FIGURE 3. DISTRIBUTION OF COUNTY PREVALENCE ESTIMATES CALCULATED BY USE OF THE DEATH RATE CONVERSION METHOD

*NUMBER IN ( ) INDICATES THE POPULATION FOR THE COUNTY. THE TOTAL REGION POPULATION IS 1,753,088 PERSONS.

# CHRONIC BRONCHITIS PREVALENCE ESTIMATION FOR TEN COUNTIES IN A HYPOTHETICAL GEOGRAPHICAL REGION



☐ 0-3000 PERSONS

☐ 3001-6000 PERSONS

▨ 6001-9000 PERSONS

■ 9001 PERSONS AND OVER

FIGURE 4. DISTRIBUTION OF COUNTY PREVALENCE ESTIMATES CALCULATED BY USE OF THE U.S. CRUDE PREVALENCE RATE ESTIMATE

*NUMBER IN ( ) INDICATES THE POPULATION FOR THE COUNTY. THE TOTAL REGION POPULATION IS 1,753,088 PERSONS.

yields the highest values. The total prevalence rate estimates for the entire region, however, are quite similar for the three methods, ranging from 2935 to 3391 per 100,000 total population.

Because the death rate conversion method is based on actual mortality data, which is converted to prevalence rate estimates by use of population weighted ratios of cases to death, this method is expected to quantify local variations in prevalence better than the other two methods. The similarity of its overall prevalence rate for the region to those of the other methods, however, indicates that over a number of samples all three methods should yield similar average rates. The estimates from the death rate conversion method will usually be lower than those from the synthetic estimation method, because of the expected tendency of this synthetic estimation model to overestimate prevalence and death rates for chronic obstructive lung disease (see above). In this example, the synthetic estimates of prevalence are quite similar to the crude estimates because the estimates have a narrow range and values similar to the crude rate. Although similar, they do provide improved sensitivity to differences in local population composition.

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# QUANTITATING THE EFFECTS OF ENVIRONMENTAL STRESS: ALTERATIONS IN DISEASE RATES

Anneke de Werk Neal

There are several approaches to obtaining quantitative information on relationships between exposure to pollutants and health effects. Toxicology studies permit the careful control of the most important variables (covariates), the utilization of a wide range of exposures, and the examination of body tissues. However, there are difficulties in extrapolating animal data to humans. Experimental or laboratory exposure of human volunteers allows the control of covariates, but a wide range of exposures and long-term exposures are not easily evaluated. Epidemiologic studies, community and occupational, investigate the health effects of complex long-term and short-term pollutant exposures on susceptible population groups. Examination of variations in disease rates or mortality rates between population groups is used to identify possible etiologic factors. Epidemiologic studies, however, employ relatively crude health measurements, must deal with a host of strong covariates and are restricted to a limited range of exposures. The assessment of adverse health effects from pollutant exposure is most effectively accomplished by using all the research approaches presented above. This discussion, however, will focus on the epidemiologic approach. Others will cover body burden and physiological changes which are concerned with toxicologic and clinical studies.

Epidemiologic investigations of air pollution health effects attempt to determine the associations between levels of specific air pollutant exposures and the rate of mortality or morbidity (disease), or the occurrence of physiologic changes in body functions within population groups. These investigations may be approached in several ways. Assuming an appropriate characterization of an area according to the expected degree of its pollution problem, comparisons of disease or death rates in urban metropolitan populations versus those in rural populations may be made. It is also possible to compare disease or death rates for migrants in their native countries with their disease or death rates in the countries to which they migrate. Such rate differences in migrating populations may be examined for their relationship with environmental changes. Another approach involves the comparison of demographic units and analysis of the relationships between disease or death rates and various pollution indices by multiple regression analysis. This regression method is a valuable statistical technique for separating the effects of different variables. The morbidity studies presented in this section employ a variation of the latter approach in which the disease rates in susceptible population groups experiencing varying urban exposures to suspect pollutants are analyzed for significant differences.

Epidemiologic studies are carried out in a real environment. It is important in such studies to consider those factors which may affect the evaluation of specific pollutant levels as well as the evaluation of human health. In the assessment of pollutant exposure levels, the following are important considerations: use of reliable methods of measurement; the chemical and physical nature of the pollutant; synergistic relationships between pollutants; determination of an adequate number of measurements for appropriate sampling times to obtain reasonably good measures of individual exposures; and meteorological conditions. In comparing the disease or death rates of people, it is necessary to evaluate disease-related factors such as smoking habits or occupational exposure to pollutants; the heterogeneous nature of the health of a diverse population; previous exposure to pollutants; medical history; nutritional status; and different climatic conditions. Unfortunately, epidemiologic studies to date have not been able to consider more than a few of the significant factors cited above. However, as more sophisticated investigative techniques become available, epidemiologic data will become more reliable.

In epidemiologic studies an association between a pollutant exposure and an adverse health effect is most likely to be causal if it supports our overall biological knowledge; if it can be demonstrated by more than one research approach; if it is consistently observed by different investigators; and if increased exposure levels are accompanied by increased frequency of the disorder. The latter is commonly referred to as a dose-response or exposure-effect relationship. The purpose of many environmental epidemiologic studies is to define a dose-response relationship for variables suspected or known to be causally associated. Not only does a dose-response effect strengthen a causal association, but a dose-response relationship can be plotted and used to quantitatively estimate the expected amount of disease and mortality associated with measured concentrations of individual pollutants. It should be mentioned here that occupational epidemiologic studies have been important in establishing dose-response relationships for many pollutants. Occupational studies generally offer a better-defined cohort, a wider range of pollutant exposures, and higher levels of exposure than community studies. However, it is difficult to apply occupational dose-response relationships to environmental evaluations because community exposures are generally 24 hours/day compared to 8 hours/day; community exposures begin at birth rather than age of employment; and the occupationally-exposed seldom include hypersusceptible population groups.

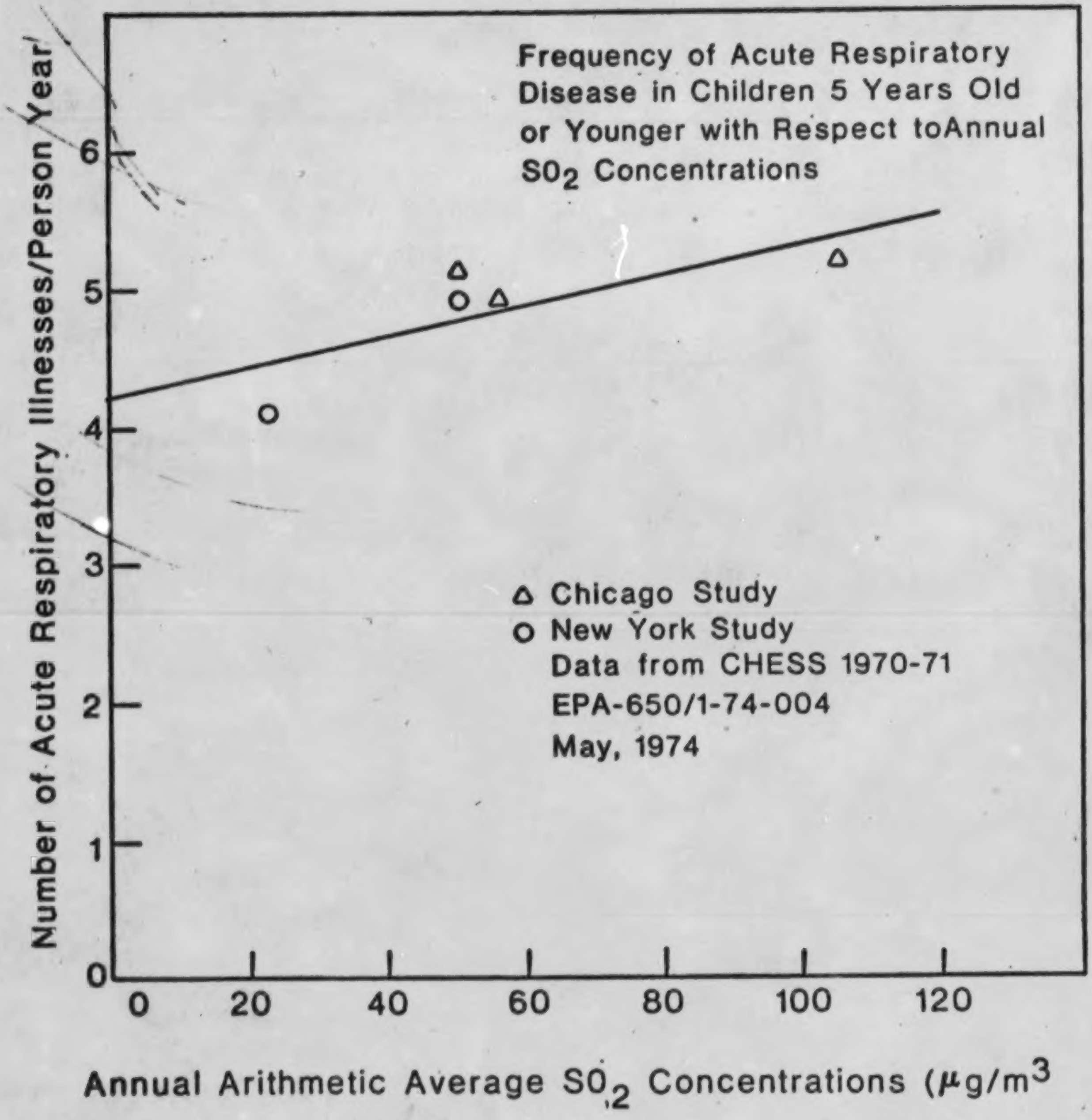
The primary purpose of this discussion is to present several existing environmental dose-response relationships which relate alterations in disease rates and mortality rates to air pollution levels. These relationships may be applied in the quantitative evaluation of the impact of energy production and use on human health. The background, assumptions and limitations of these relationships will also be discussed.

Specific dose-response relationships are not known for most pollutants. As a result, the pollutants included in this section are limited to sulfur dioxide ($SO_2$), suspended sulfates, and total suspended particulates (TSP). Sulfur dioxide is known to be a respiratory irritant. The irritant effect of $SO_2$ results from the conversion of $SO_2$ to sulfurous acid in the presence of water at the membranes. When $SO_2$ is experienced in association with particulates, the $SO_2$ may be adsorbed onto the surfaces of respirable aerosols and carried into the lower respiratory tract where the droplets can be transformed into more highly irritant substances such as sulfuric acid and metallic sulfates. It is believed that all atmospheric $SO_2$ is eventually oxidized to sulfates either in air or after absorption by plants or adsorption onto surfaces (7). The toxicologic action of particulate matter may also occur in other ways: 1) the particle may be intrinsically toxic, 2) the particle may interfere with clearance mechanisms in the respiratory tract, and 3) the particle may act as a carrier of toxic material.

## SULFUR DIOXIDE: ACUTE RESPIRATORY ILLNESS IN CHILDREN

Figure 1 depicts the relation between annual arithmetic averages of $SO_2$ and the number of acute respiratory illnesses expected per person per year for children five years or younger. Acute respiratory illness is an important multibillion dollar public health problem identified as the leading cause of acute morbidity in the U. S. (4). Children experience a much higher rate of incidence of acute respiratory disease than the population at large and hence have been the focus of a number of environmental epidemiologic studies. Two such studies, prospective surveys in New York (5) and Chicago (4) of

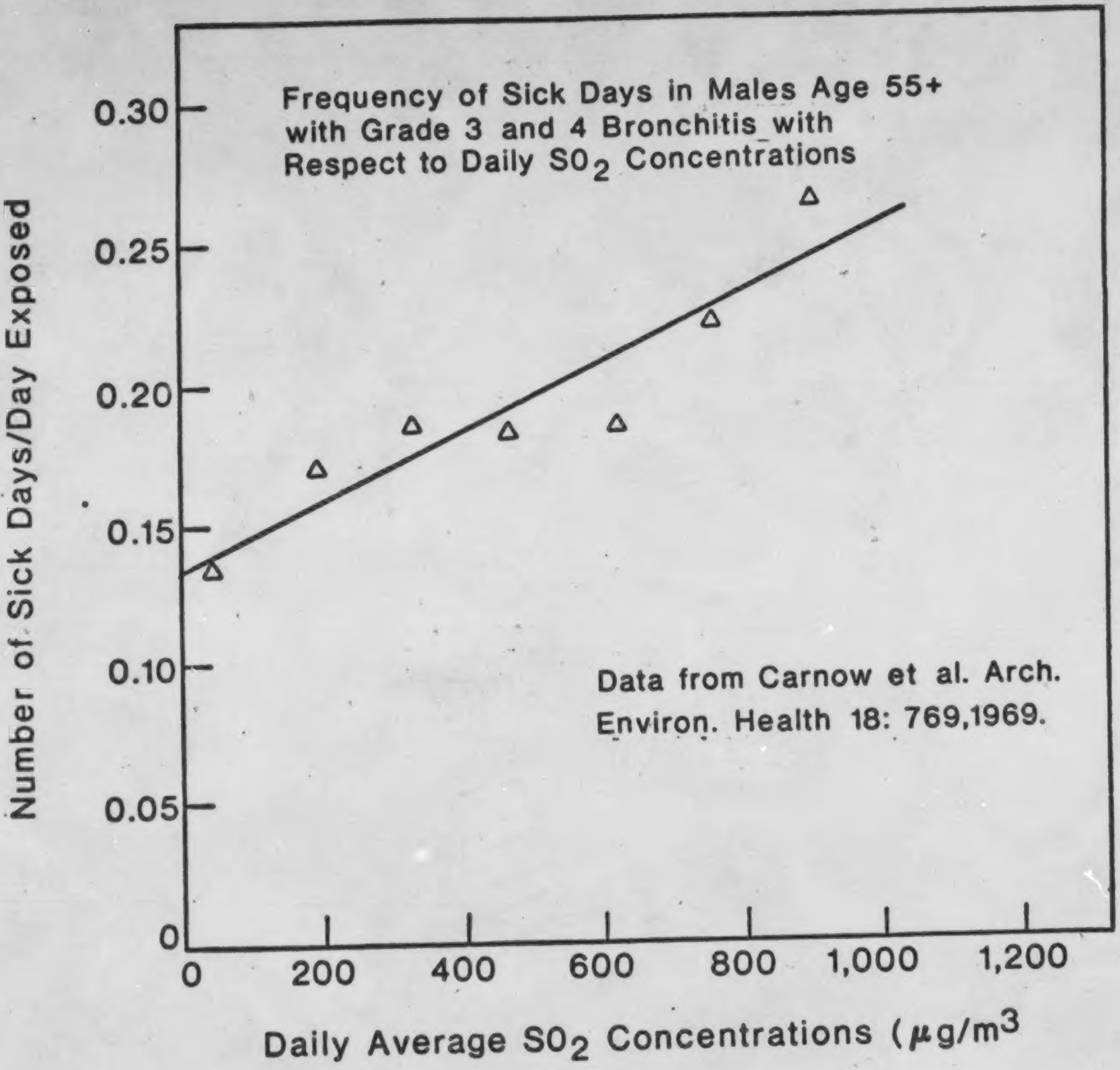
Figure 1.


Frequency of Acute Respiratory Disease in Children 5 Years Old or Younger with Respect toAnnual $SO_2$ Concentrations
Number of Acute Respiratory Illnesses/Person/Year
0
1
2
3
4
5
6
0
20
40
60
80
100
120
Annual Arithmetic Average $SO_2$ Concentrations ($\mu g/m^3$
△ Chicago Study
○ New York Study
Data from CHESS 1970-71
EPA-650/1-74-004
May, 1974

acute respiratory disease conducted through the CHESS program, provide the basis for Figure 1. These studies were combined because the data were collected, analyzed and presented in a similar manner, the subjects were similar in socioeconomic and other factors, and because the pollution mix of the two cities were fairly comparable. The attack rates of acute respiratory illnesses in children were obtained by telephone interviews with the mothers. The illness rate includes both upper and lower respiratory diseases. The Chicago data (over 600 children) were collected over a period of 46 weeks (1969-70). The New York study (300 children) lasted 30 weeks (1970-71) and both study periods included an influenza season. The rates in Figure 1 have been adjusted to extend over a period of one year. Because the studies ran during seasons with highest illness rates, extrapolation of rates to one year may be a little high. All children in both studies resided within 1.5 miles of an air monitoring station. The children in Chicago were divided into three residential pollution exposure categories as measured by the air monitoring stations during 1969-70. The annual arithmetic average $SO_2$ concentration in the intermediate exposure area was 57 $\mu g/m^3$ (with 111 $\mu g$ $TSP/m^3$); in the high area it was 51 $\mu g/m^3$ (with 126 $\mu g$ $TSP/m^3$); and in the highest, it was 106 $\mu g/m^3$ (with 151 $\mu g$ $TSP/m^3$). The New York children resided in a low-pollution area where the annual average $SO_2$ concentration was 23 $\mu g/m^3$ and annual average TSP level was 34 $\mu g/m^3$, or they resided in an intermediate pollution area with annual averages of 51 $\mu g$ $SO_2/m^3$ and 63 or 86 $\mu g$ $TSP/m^3$.

## SULFUR DIOXIDE: CHEST ILLNESS IN BRONCHITICS

Figure 2 shows the relationship between daily average $SO_2$ levels and expected number of sick days (chest illness) per day of exposure for males aged 55 and over with chronic bronchitis grades 3 and 4. This figure is based on results from a study which was designed to investigate the dose-effect relationship between air pollution and morbidity from respiratory disease in patients with chronic broncho-



Frequency of Sick Days in Males Age 55+ with Grade 3 and 4 Bronchitis with Respect to Daily $SO_2$ Concentrations

Data from Carnow et al. Arch. Environ. Health 18: 769,1969.

Figure 2.

pulmonary disease (2). An increase in the acute illness rate with increased levels of $SO_2$ was found for males 55 years and over with grade 3 and 4 bronchitis. These results are depicted in Figure 2. The data points shown represent the illness days of over 100 volunteer bronchitics from October 1, 1967 through November 30, 1967, as determined from patients' daily responses on a chest illness calendar. Chest illness or sick day was defined as one on which a patient was bothered much more than usual by his chest for most of the day, had more cough than usual, suffered more shortness of breath than usual, was less active than usual because of his chest, or had a chest cold. A grade 3 bronchitic was defined as one who coughed or raised phlegm throughout the day and year for more than two years, and a grade 4 bronchitic had to experience these symptoms for more than five years. Daily average $SO_2$ concentrations in each patient's square mile of employment or residence were estimated, based on $SO_2$ measurements made at eight city-wide continuous monitoring stations. The plot in Figure 2 was derived by dividing the number of sick days per person occurring one day after exposure to a particular pollution concentration i by the number of days per person exposed at level i. The selection of the fall study period (as opposed to winter) avoided the problem of severe low temperatures complicating health effects and the problem of bronchitics experiencing less outdoor exposure during the winter.

## SULFUR DIOXIDE: MORTALITY

Figure 3 is a graph showing a stepwise dose-response relationship between mortality and $SO_2$ concentrations. This figure represents the results of a study (1) which evaluated daily variations in the death rate for the entire New York metropolitan area for 1962-1966 in relation to the air quality data as measured at one monitoring station. The application of air quality data from one station to an entire metropolitan area renders this relationship as rather impre-

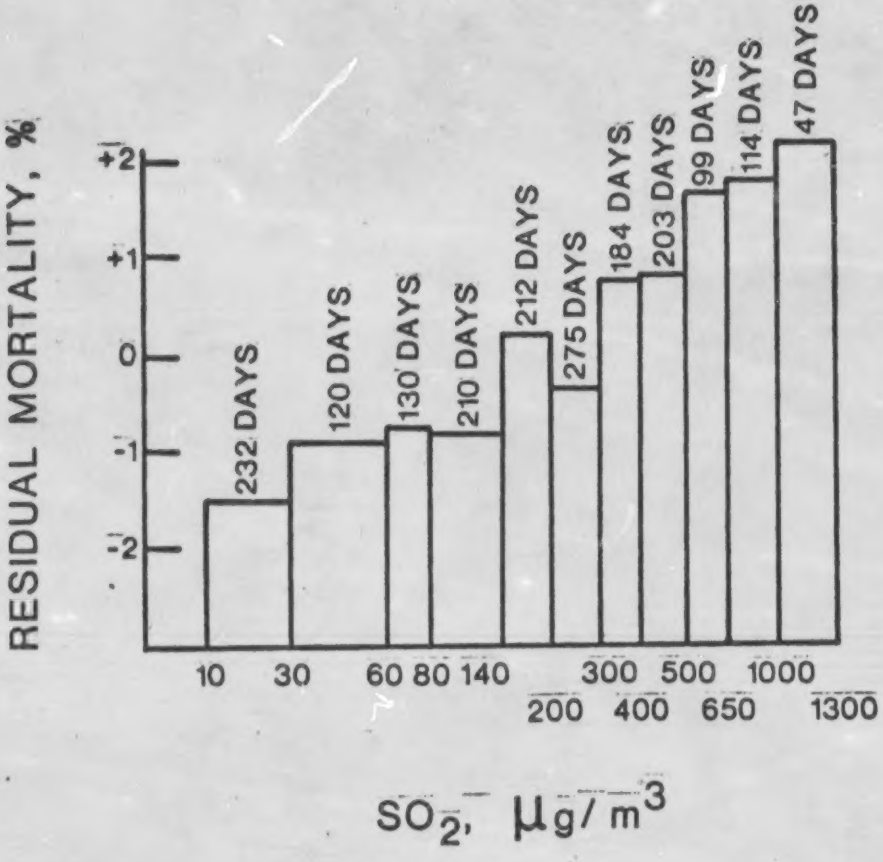


FIGURE 3. MEANS OF RESIDUAL MORTALITY BY $SO_2$ LEVELS, 1962 TO 1966 (1,826 TOTAL DAYS)(1)

cise, even though the investigators adjusted the death rates for seasonal cycles, temperature fluctuations and other confounding variables. Figure 3 indicates that on days when $SO_2$ levels were above 500 $\mu g/m^3$ (0.19 ppm) mortality was approximately 2% greater than expected. For days when $SO_2$ concentrations were below 30 $\mu g/m^3$ (0.01 ppm) mortality was 1.5% less than expected.

## SUSPENDED SULFATES: ASTHMATICS

Figure 4 presents temperature-specific dose-response relationships for suspended sulfate levels and resultant asthma attack rates in New York. These data were obtained during the CHESS study of aggravation of asthma by air pollutants. The New York study (3) was chosen because the environmental pollutants in New York are more similar to those of Chicago than those of the other study sites. A panel of 148 asthmatics recorded asthma attacks on a daily diary from October 1970 through May 1971. All subjects lived within 1.5 miles of a monitoring station and were divided among three communities of differing air quality. Average daily suspended sulfate concentrations were obtained by analyzing total suspended particulates for the sulfate fraction using a turbidimetric method. The resultant dose-response function plotted in Figure 4 predicts that a suspended sulfate exposure of 35 $\mu g/m^3$ would be associated with a 4% increase in asthma attacks on cooler days (>30°F and <50°F) and a 57% increase on warmer days (> 50°F). When using these relationships, it should be recognized that the study did not include the effect of plant allergens nor the potential effect of other pollutants.

## TOTAL SUSPENDED PARTICULATES: MORTALITY FROM CHRONIC RESPIRATORY DISEASE

Figure 5 relates the average annual death rate from chronic respiratory disease with particulate pollution levels measured in the

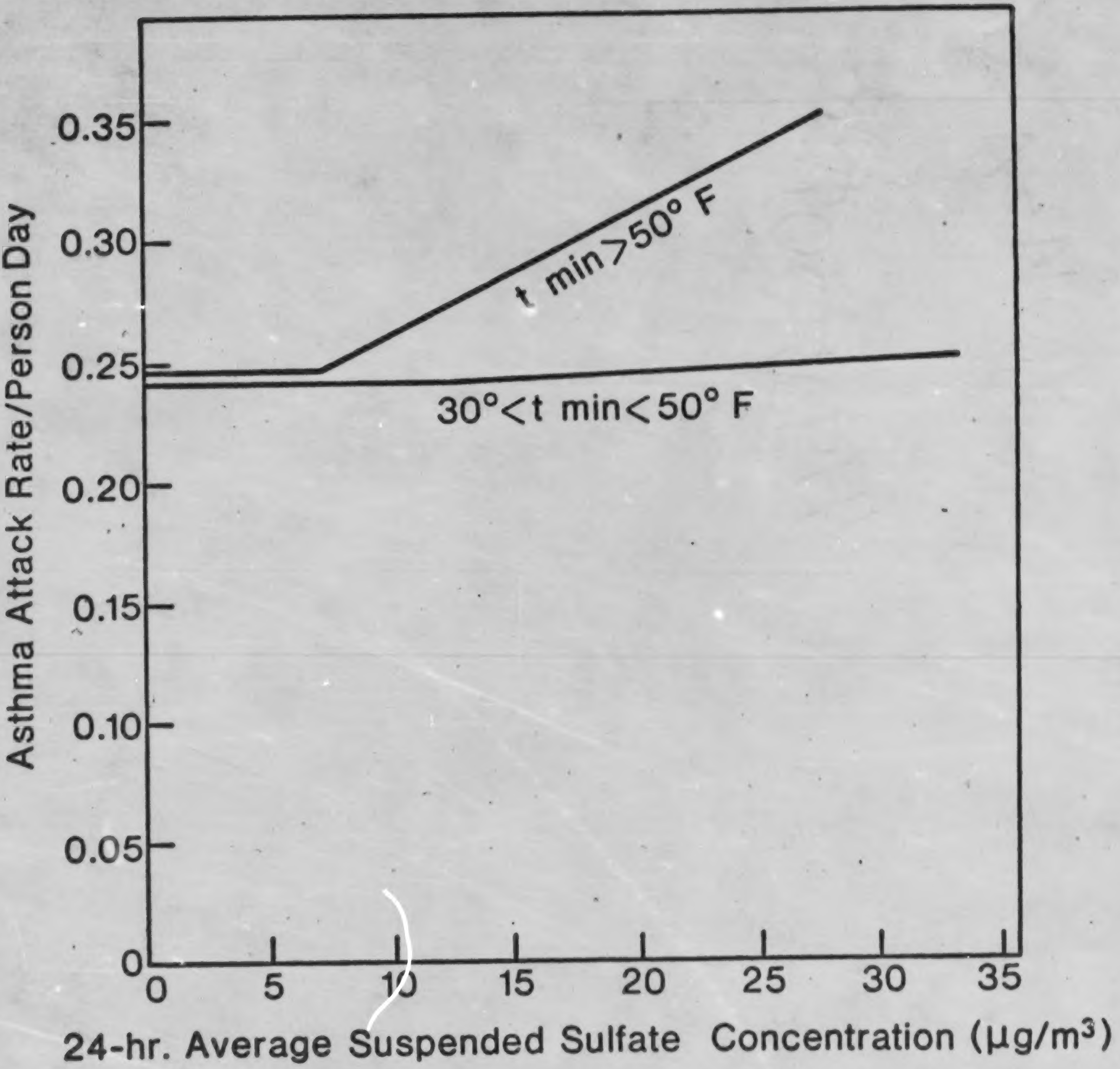
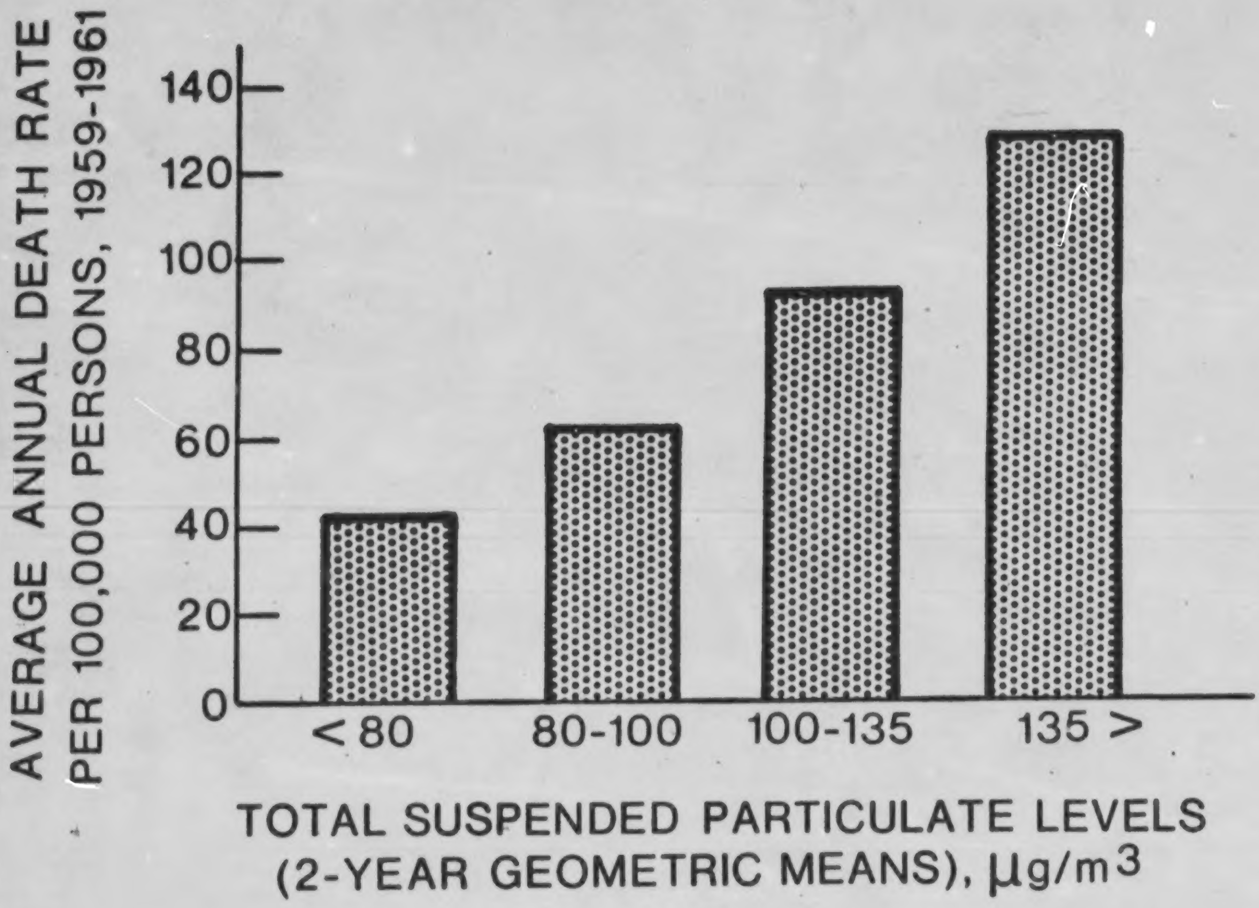


Figure 4. Asthma attack rate versus suspended sulfate concentration (3).

Fig. 5 AVERAGE ANNUAL DEATH RATE FROM ASTHMA, BRONCHITIS OR EMPHYSEMA, FOR WHITE MALES (AGE 50-60), VERSUS LEVELS OF PARTICULATE MATTER.



Buffalo area (8-10). A strong correlation was found between chronic respiratory disease mortality in white males 50-69 years old and suspended particulate levels, regardless of the economic level of the deceased. Total suspended particulate concentrations were measured at 21 air sampling stations in and around Buffalo from July 1961 until June 1963. These data resulted in the designation of four particulate exposure areas: the low-exposure area had a 2-year geometric mean of under 80 $\mu g$ TSP/$m^3$; Level 2 had 80-100 $\mu g/m^3$; Level 3 had 100-135 $\mu g/m^3$; and Level 4 was greater than 135 $\mu g/m^3$. Mortality data for the study area were obtained for the period of 1959-1961 using the 1960 census as the population basis. Figure 5 indicates that the death rate from chronic respiratory disease was 44 per 100,000 persons in the lowest pollution area, almost 40% higher for persons in the next most polluted area, and approximately three times as high for persons in the highest pollution area.

In this study no consideration was given to occupation. Other limitations of this type of analysis include the inability to obtain information on the deceased's smoking habits and the uncertainty that the pollution levels are representative of exposure at the time the chronic disease conditions were developing.

## GRAIN DUST (SUSPENDED PARTICULATES): ASTHMA INCIDENCE

A University of Minnesota study investigated the relationship between air pollution generated by grain handling and processing facilities and allergic diseases (6). Grain sensitive students were found to experience a statistically significantly higher incidence of bronchial asthma attacks on days when the grain air pollution was high (when mean soiling index was greater than 0.25 COH) and the average wind direction was from a large grain plant complex to student housing areas, than on all other days. These results are displayed in Table I. Although the data are limited, they do provide a basis for

### Table 1. Asthma Incidence Among Students During Conditions Favorable And 'Less Favorable For Exposure To Grain Dust (6)

| Groups [a] | Less Favorable [b] (157 days) | Favorable [c] (68 days) |
|---|---|---|
| Grain sensitive | 0.0320 | 0.0520 [d] |
| Grain negative (control) | 0.0280 | 0.0340 [e] |

(a). 126 students with history of allergic bronchial asthma were divided into grain positive (51) and grain negative (75) subgroups based on allergic skin test reactions

(b). All other days

(c). Days when mean soiling index > 0.25 COH and average wind direction from source to student residences

(d). S.E.D. : ± 0.0062, $p = 0.001$

(e). No significance

a dose-response evaluation which combines pollution concentrations and wind direction.

The above examples represent some of the more recent and more reliable dose-response relationships which may be used quantitatively. The limitations of these studies should be recognized when such relationships are applied. However, they do provide a means for, at least, preliminary quantitation of human health effects of pollutant exposure. As more pollution exposure information and community health data become available, better dose-response relationships will become available.

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# QUANTITATING THE EFFECTS OF ENVIRONMENTAL STRESS: BODY BURDEN

A. L. Miller

Body burden is the amount of element or compound in the body at any time, usually expressed in units of weight, e.g., μg, (or for radioactive material, μCi). The total body burden at any time is due to the sum of exposures from many sources such as hobbies, food, dishes and the environment (ambient and occupational). Total body burden is the sum of the burdens for all the individual organs; one or more of which may be considered as critical organs.

A critical body organ is that organ of the body whose damage by the radiation or pollutant results in the greatest damage to the body. It is readily apparent that many factors must be considered in determining which affected organ will cause the body to suffer the the greatest damage.

The criteria of prime importance in determining which organ is critical (1) are:

a. The organ that accumulates the greatest concentration of the material;

b. The essentialness or indispensability of the organ to the well-being of the entire body;

c. The organ damaged by the route of entry of the material into the body;

d. The sensitivity of the organs, i.e., the organ damaged by the lowest dose.

See Figure 1 for some examples of toxic agents and their respective critical organs.

## FIG. 1 CRITICAL ORGANS AND RELATED TOXIC AGENTS

| Organ Systems | Toxic Agents |
|---|---|
| BRAIN | Methyl mercury;Organic lead; Carbon Monoxide; Mercury;Manganese |
| NASOPHARYNGEAL | Cadium; Arsenic; Nickel; Chromium |
| LUNGS | Manganese; Magnesium; Beryllium; Zinc, Asbestos; Sulfur Oxides; Ammonia; Cadmium; Ozone; Nitrogen Oxides, Peroxyacetyl Nitrate; Nickel Carbonate; Hydrogen Sulfide |
| LIVER | Selenium<br>Chlorinated Hydrocarbons |
| KIDNEY | Mercury; Cadmium |
| BONE | Flouride; Lead; Strontium-90; Radium; Plutonium |
| SKIN | Arsenic; Beryllium; Nickel; Chromium |
| FAT | Chlorinated Hydrocarbons (e.g., DDT) |
| GASTRO-INTESTINAL | Zinc; Lead; Arsenic; Fluoride; Vanadium |
| THYROID | Cobalt; Iodine - 131 |
| HEART | Carbon Monoxide |

Two prime factors that affect body organ burden are the environment to which the individual is exposed and the characteristics of the individual. The two principal components in the body burden relationship due to exposure in the environment are uptake and elimination. Uptake can basically be defined as follows:

Uptake = Intake x fraction retained or deposited

Intake, the amount inhaled or ingested per unit of time, e.g., µg/day, is directly related to the concentration in the environment (air, water and food). Therefore, the concentration of the material in the environment is one of the principal factors of concern. Body burden estimates are usually based on "reference man." Due to anatomical and physiological differences among individuals, mean values for such things as body weight, blood composition and volume are used for such calculations. For example, reference man weighs 70 kg and has a liver weight of 1.8 kg (2). Reference man inhales about 20 $m^3$ of air/day and ingests about 2.2 liters of water/day . The amount of material inhaled or ingested per day may be estimated in the following manner:

Total ingested = mass from water + mass from food
= (volume of water ingested) x (concentration in water)
+ (mass of food) x (concentration in food)

Total inhaled = mass from air
= (volume inhaled) x (air concentration)

Total intake = total ingested + total inhaled

One may inhale and/or ingest the same material from many sources. Each of these sources contributes to the total body burden. In addition, the nature of the material must be considered, i.e., is it in a gas, vapor or particulate form?; what is its chemical form?; its solubility in body fluids? If it in particulate form, the particle size distribution must be considered in determining the amount of the dust that can penetrate into the various regions of the lung.

Even when the environmental exposure is the same, two individuals may have greatly differing body burdens. This difference may be attributed in a large part to differences in age and sex of the individuals; level of physical activity; presence of respiratory disease; and nutrition.

## MODELS FOR ESTIMATING BODY BURDEN

There are two rather simplified models that have been utilized extensively for estimating body burdens. These have been used particularly by the International Commission on Radiological Protection (ICRP) (1). These models are: (a) Continuous exposure to a constant concentration of a specific material with exponential elimination from a single compartment; and (b) Single intake of a specific material over a short period of time (hrs) with exponential elimination from a single compartment. When an element is ingested or inhaled it may be deposited in one or more organ. If more than one organ is involved this would represent more than one compartment. Generally then, a compartment may be considered to be an organ or organ system.

### Definition of Terms

$m$ = mass of organ

$C$ = mass concentration of pollutant in organ, material in organ/g of organ

$mC$ = body or organ burden

$f_a$ = fraction of pollutant inhaled that reaches the organ of reference

$f_w$ = fraction of pollutant ingested that reaches the organ of reference

$I$ = average amount inhaled or ingested per day, g/day

$f_2$ = fraction of total body burden that is present in organ of reference (note that $\Sigma f_2$ (for all organs) = 1.0)

$T_b$ = biological half-life, time required for half of material to be eliminated from organ or body

$T_e$ = effective half-life for radioactive materials

$k$ = $0.693/T_b$, fraction eliminated per unit of time

Continuous Exposure

The following is an example of application of the model for continuous exposure (for ingestion only) at constant concentration with exponential elimination from a single compartment:

Time rate of change of mass of element in organ = rate of uptake from ingestion - rate of elimination

$$\frac{d(mC)}{dt} = If_w - k(mC) \tag{1}$$

Figure 2 illustrates a single compartment model. Integrating equation 1 gives:

$$mC = \frac{If_w}{k}(1-e^{-kt}) \tag{2}$$

where t is time of exposure. Substituting the definition of k into equation 2 gives:

$$mC = \frac{If_wT_b}{0.693}(1-e^{-0.693\,t/T_b}) \tag{3}$$

Furthermore, if $t/T_b >> 1$, then equilibrium between uptake and elimination exists, $\frac{d(mC)}{dt} = 0$,

and

$$mC_\infty = \frac{If_wT_b}{0.693} \tag{4}$$

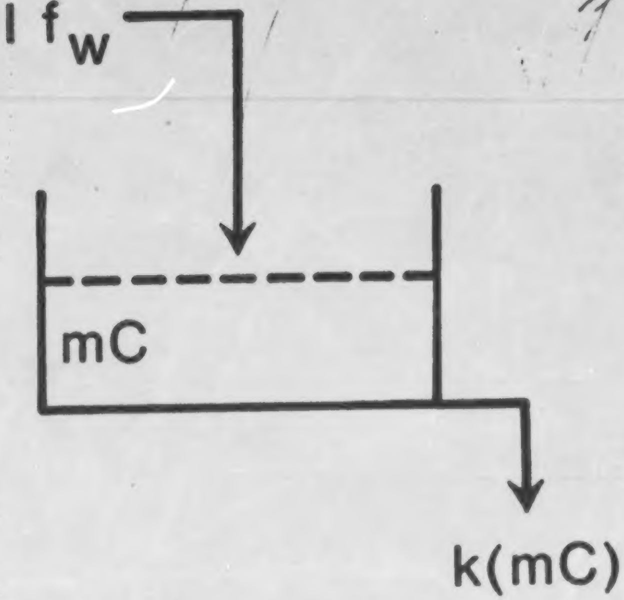


Figure 2. Single compartment model schematic

Figure 3 is a plot of equation 3. For inhalation instead of ingestion, replace $f_w$ by $f_a$ in equation 3 and 4. For insoluble material in the lung; $T_b$ = 120 days (except for plutonium for which $T_b$ = 1 year and thorium for which $T_b$ = 4 years); for radioactive material, replace $T_b$ with $T_e$.

Example of Continuous Intake of a Constant Concentration.

Assume an individual is ingesting water that contains 10 μg/liter of lead and that the following values are applicable (2) :

mass of total body = $7 \times 10^4$ g
mass of bone = $7 \times 10^3$ g
biological half-life, $T_b$ = $1.46 \times 10^3$ days (whole body)
$3.65 \times 10^3$ days (bone)

Bone as critical organ:

$f_w = 0.02$

$f_a = 0.08$

for ingestion;

$$mC_\infty = \frac{I f_w T_b}{0.693}$$

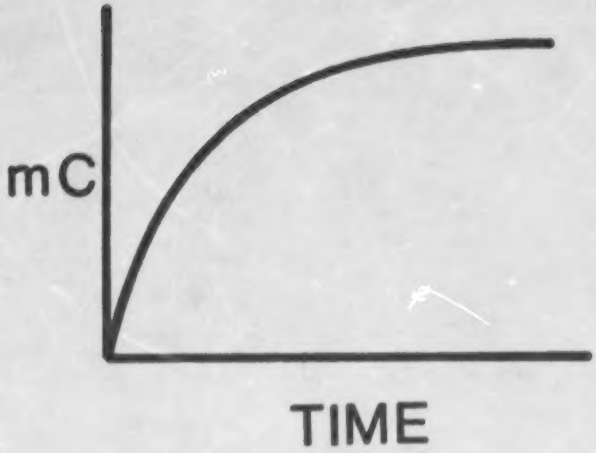
water intake = 2.2 liter/day
I = 10 μg/liter x 2.2 liter/day = 22 μg/day
$T_b = 3.65 \times 10^3$ days

Therefore:

$$mC_\infty = \frac{(22\ \mu g/day)(0.02)(3.65 \times 10^3\ days)}{0.693}$$

$mC_\infty$ = $2.32 \times 10^3$ μg or 2.32 mg of lead from water

Figure 3. Change in body burden with time for continuous exposure to a constant concentration model.



If, in addition, the individual is inhaling air with a lead concentration of 3 $\mu g/m^3$, then the body burden from inhalation will be:

$$mC_\infty = \frac{If_aT_b}{0.693}$$

air inhaled = $20\ m^3/day$

Now

$$I = 3\ \mu g/m^3 \times 20\ m^3/day = 60\ \mu g/day$$

$$mC_\infty = \frac{(60\ \mu g/day)(0.08)(3.65 \times 10^3 days)}{0.693}$$

$$mC_\infty = 25.3 \times 10^3\ \mu g = \underline{25.3\ mg} \text{ from air}$$

Total $mC_\infty = mC_\infty(\text{air}) + mC_\infty\ (\text{water}) = 25.3 + 2.32 = \underline{27.6\ mg}$ lead

Single Intake

The single intake model can be described as follows:

Initial mass of element in organ = (Amount ingested or inhaled)(fraction of the amount ingested or inhaled which is deposited in the critical organ)

$$mC_o = A_0f_w \text{ (for ingestion) or } A_0f_a \text{ for inhalation}$$

where

$C_o$ = concentration of element initially in organ (t = 0)

$A_0$ = amount of the single intake ingested or inhaled, g

For single compartment exponential elimination, the mass of element in the organ will decrease exponentially. This relationship is shown in Figure 4 and can be expressed as:

$$mC = mC_O e^{-kt} \tag{5}$$

where

$t$ = time after exposure

Examples of Single Intakes

*Ingestion*

Assume an individual ingested (e.g., by drinking from a lead glazed cup) 100 µg of lead. The amount deposited in the bone would be about:

$$mC_O = A_O f_w = 100\ \mu g(0.02) = \underline{2\ \mu g} \text{ of lead}$$

*Inhalation*

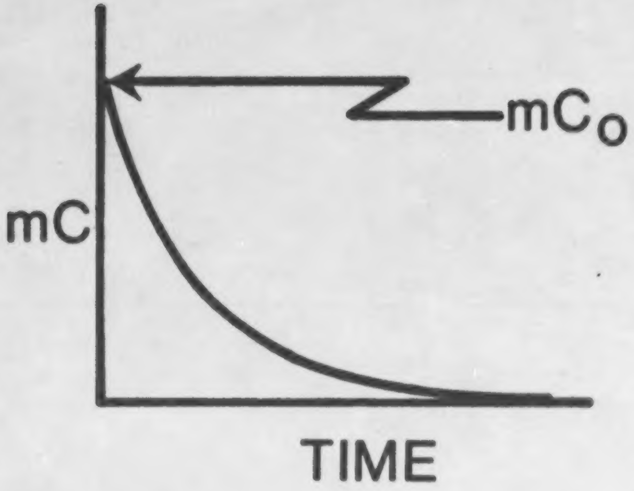
Assume an individual inhales air containing 100 µg/m$^3$ for 1 hour at the rate of 10m$^3$ for 8 hours, then;

$$A_O = 100\ \mu g/m^3 \times \frac{10m^3}{8\ hr} \times 1\ hr = 125\ \mu g$$

and:

$$mC_O = 125\ \mu g\ (0.08) = \underline{10\ \mu g} \text{ of lead}$$

Figure 4. Exponential elimination for a single intake body burden model.



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# QUANTITATING THE EFFECTS OF ENVIRONMENTAL STRESS:
# PHYSIOLOGICAL CHANGES

W. H. Hallenbeck, S. J. Jansen and W. H. Kojola

Until recently an environmental pollutant was not considered a threat to human health unless its concentration was high enough to produce an observable disease syndrome. An observable disease syndrome or symptom is referred to as a clinical effect. Recent evidence has shown that relatively small physiological changes are produced by low concentrations of environmental pollutants. Relatively small physiological changes which do not produce an observable disease syndrome or symptom are referred to as subclinical effects. Subclinical effects range from subtle changes in blood chemistry which may be reversible to small changes in nervous tissue which may be irreversible and permanent. An important characteristic of a subclinical effect is that it is usually below human perception and can only be detected by biochemical or physiological tests. However, such effects do represent interruption of normal physiological processes.

Subclinical effects raise at least two important health questions. Even though a healthy person does not perceive a pollutant-induced subclinical effect, is there a cumulative effect from chronic low-level exposure which may result in a clinical effect? Little is known about this question and much research is needed. Secondly, are there hypersusceptible human population groups for whom a small physiological change may trigger a clinical reaction? There is evidence which clearly indicates that certain groups are at risk to reacting adversely to concentrations of pollutants which do not perceptibly affect healthy persons. Two such high-risk groups are persons with pulmonary and/or cardiovascular disease.

Many variables enter into the process of planning new energy producing and using facilities. A variable which deserves special consideration is the health implications of the facility for hypersusceptible population groups. In addition, as research provides more information, it may become important to consider the effect of long-term exposure to pollutant concentrations which produce subclinical effects in healthy persons. Carbon monoxide, lead and ozone are three pollutants which produce subclinical effects. The following discussion of these pollutants illustrates the nature of subclinical effects due to environmental stressors on healthy and hypersusceptible groups.

## PHYSIOLOGICAL CHANGES DUE TO CARBON MONOXIDE

Exposure to carbon monoxide (CO) in the environment or workplace results in physiological changes which reduce the oxygen-carrying capacity of the blood. Depending on the ambient concentration of CO, the length of exposure, and individual susceptibility, these physiological changes may result in subclinical or clinical health effects.

Carbon monoxide diffuses rapidly across the alveolar membranes in the lung and binds reversibly with hemoglobin (Hb) in the red blood cells to form carboxyhemoglobin (COHb) (Fig. 1).* The affinity of Hb for CO is 200-250 times that for oxygen ($O_2$) (33). In addition to COHb formation, exposure to CO causes $O_2$ to bond more tightly with the remaining Hb. The increased tightness of the oxyhemoglobin ($HbO_2$) bond means that tissue oxygen must fall to lower than normal levels before $HbO_2$ gives up its $O_2$ (7).

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*Coburn, Foster, and Kane (CFK) have developed a theoretical equation which relates exposure to CO to percent COHb saturation (Fig. 1) (14). Data obtained from human exposure studies on healthy young adults strongly support this model (30). Note in Figure 1, that the rate of attainment of COHb equilibrium increases as the concentration of CO increases. The primary ambient air standard for CO is 9 ppm for 8 hours and 35 ppm for one hour.

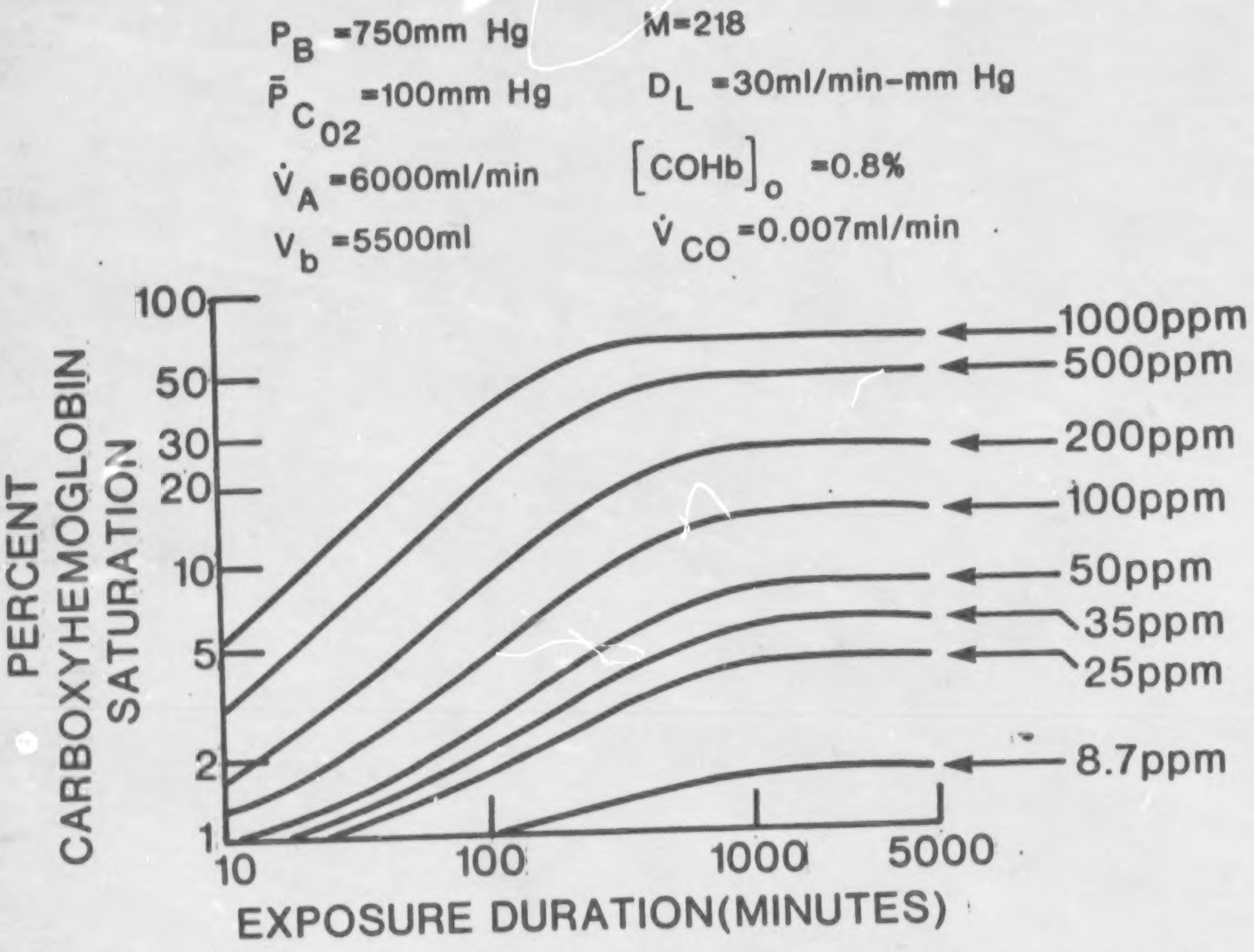

Fig.1 Carboxyhemoglobin levels for man as a function of exposure duration and the CO concentration as determined by solving the CFK equation (30).

The presence of CO in the human body may originate from endogenous and exogenous sources. Normal breakdown of Hb produces a low concentration of endogenous CO which results in a COHb saturation of 0.3-0.7%. Disease states may contribute to endogenous CO production. For example, patients with hemolytic anemia may have a COHb saturation of 4-6% (33).

Exogenous sources of CO arise from the incomplete combustion of carbonaceous material. Tobacco smokers are the most heavily exposed nonindustrial group and may have a COHb saturation of 6-9% (33). The increased COHb levels caused by smoking cigarettes have been shown to decrease the rate of oxygen delivery to heart tissue with subsequent reduction in the ability to exercise before onset of angina pain (5). Motor vehicle exhaust accounts for about 80% of annual CO emissions. Peak-hour traffic can generate 25 ppm of CO and, during inversions, may rise to 100 ppm. Fuel combustion at stationary sources, industrial processes, and solid waste disposal account for another 20% of the total emissions of CO (33).

Myocardial (heart muscle) tissue has a high demand for $O_2$. At rest, 75% of the $O_2$ is extracted from the coronary circulation as compared to 25% in peripheral tissue. Normally, increased myocardial demand for $O_2$ is met by increasing the flow rate of coronary circulation. Consequently, a healthy person is able to adapt to a modest increase in COHb. However, coronary vascular disease may prevent an increase in blood flow to compensate for stresses such as COHb, a tightened $HbO_2$ bond, and exercise (3,7). In this event, an alternative compensating mechanism comes into play. Myocardial tissue will extract $O_2$ from the blood at a greater rate; increased $O_2$ extraction may reduce $O_2$ in the blood to low levels. If low enough blood levels are reached, some areas of the heart muscle will be oxygen starved and subclinical to clinical myocardial necrosis may result in persons with heart disease. Aronow et al. (3) have observed the effects of a 90-minute exposure of ten angina patients to about 53 ppm of CO in

Table 1. Human Response to Various Carboxyhemoglobin Levels (33).

| CO ppm | Time of exposure hours | % COHB saturation | Response of healthy adult | Response of patient ill with severe heart disease (hypersusceptible) |
|---|---|---|---|---|
| | | 0.3-0.7 | Endogenous CO production. No known detrimental effect. | |
| | | 1.5 | Increase in blood flow to certain vital organs such as the heart. | May lack sufficient cardiac reserve to compensate. |
| 50 | 1.5-4 | 2.7-5.1 | Visual light threshold increased. | Less exertion required to induce pain in patients with angina pectoris (2-4). |
| | | 16-20 | Headache, visual evoked response abnormal. | May be lethal. |
| | | 20-30 | Throbbing headache; nausea; fine manual dexterity abnormal. | |
| | | 30-40 | Severe headache; nausea and vomiting; syncope. | |
| | | 50 | Coma; convulsions. | |
| | | 67-70 | Lethal if not treated. | |

Los Angeles freeway air. The patients developed a mean increase in COHb levels of 4% and their ability to exercise before onset of angina was significantly decreased. Average COHb levels increased from 1 to 5% before and after exposure. Anderson et al. (2) demonstrated that a mean increase in COHb of only 1.6% can cause significantly decreased exercise tolerance in persons with stable angina pectoris; 10 patients were exposed to 50 ppm of CO under laboratory conditions for four hours. Average COHb levels increased from 1.3-2.9% before and after exposure to CO. Aronow and Isbell (4) exposed 10 patients with exertional angina pectoris to 50 ppm CO for two hours under laboratory conditions. A mean increase in COHb of 1.7% caused significantly decreased exercise tolerance. Average COHb levels increased from 1.0-2.7% before and after exposure.

Table 1 lists some of the known subclinical and clinical physiological responses to CO exposure. Whether clinical or subclinical health effects follow exposure to ambient CO depends largely on individual susceptibility. The following groups are hypersusceptible to the toxic effects of CO: heavy smokers, infants, and patients with cardiovascular disease, anemia, lung disease, and increased metabolic rate (33). Individuals with hemolytic anemia or who are heavy smokers will have elevated COHb levels before exposure to ambient CO. Persons with severe heart disease may not possess sufficient cardiac reserve to compensate for CO exposures to which healthy members of the population can adapt.

## PHYSIOLOGICAL CHANGES DUE TO OZONE

Ozone is one of the major environmental air pollutants present in the breathable atmosphere, with concentrations ranging from less than 0.05 ppm to as much as 0.70 ppm in the urban environment (34). Ozone is recognized as a bronchopulmonary irritant which adversely affects human mucous linings and lung tissue as well as normal respiratory functioning (34). When inhaled it initiates biochemical changes in the blood (11). While ambient levels of ozone have not, as yet, been unambiguously linked to the production of disease states in humans, there is evidence that these levels may result in physiological and biochemical changes which constitute subclinical health effects.

Perturbations in several lung physiology parameters (including reductions in vital capacity, expiratory flow rates and diffusing capacity, along with increases in pulmonary resistance) are the predominant subclinical health effects resulting from ozone inhalation. Humans with normally functioning lungs exhibited subclinical lung changes at 0.50 ppm ozone after two-hour exposures on two consecutive days (19,20). Exposure of normals at 0.75 ppm for a single two-hour period also caused significant decrements in lung function (8,38). Exposure of normals to 0.90 ppm for as little as five minutes immediately induces significant changes in these measures of lung physiology (23). In addition to the subclinical effects present during the above-mentioned ozone concentrations, individuals also reported symptoms of dry cough, tracheal irritation and soreness in the chest following inhalation.

Human populations hypersusceptible to ozone exposure will exhibit these pulmonary subclinical changes at ozone levels below concentrations which begin to produce similar effects in normals. Humans with normal lung function but classified as having "pulmonary hyperreactivity" (a history of cough and chest discomfort associated with allergy or air pollution exposure) will develop significant pulmonary function changes beginning at 0.37 ppm, with or without exercise (19). Individuals with severe pulmonary emphysema are another hypersusceptible group. After inhaling ambient ozone between 0.20-0.53 ppm for three to four consecutive days, such individuals exhibited pulmonary physiological changes which are reversible by breathing filtered air for 40 hours or more (26).

Table 2 summarizes the lung physiology changes following ozone exposure and indicates the level of exposure, the type of subject exposed (normal or hypersusceptible) and whether the changes are statistically significant.

Subclinical biochemical changes in blood also occur in humans inhaling ozone. Biochemical analyses reveal that several changes in red blood cell and plasma enzymes begin to appear at 0.25 ppm as well as an increase in red cell membrane fragility. The enzyme changes include increases in glucose-6-phosphate dehydrogenase and lactate dehydrogenase activities and decreases in red blood cell acetylcholinesterase activity and reduced glutathione levels. These biochemical changes become statistically significant at 0.37 and 0.50 ppm ozone exposure. Such alterations disappear gradually but are still detectable two weeks after inhalation (10,11).

In conclusion, exposure to ozone at concentrations present in the breathable atmosphere will produce subclinical physiological and biochemical changes in humans. These changes occur in hypersusceptible individuals (those with pulmonary emphysema or pulmonary hyperreactivity) at ozone levels lower than those which produce similar effects in normals.

## PHYSIOLOGIC CHANGES DUE TO LEAD

Although cases of clinical lead poisoning in children and workers remain a problem today, the question of subclinical physiologic effects at low levels of exposure is of increasing concern. A few examples of such potential effects will be discussed to illustrate the reason for concern.

Lead can interfere with the manufacture of heme and iron utilization. Heme is the precursor of hemoglobin which is the $O_2$ carrying pigment

Table 2. Normal and Hypersusceptible Human Responses to Ozone Inhalation

| Ozone Level (ppm) | Duration of Exposure | Type of Subject | Exercise | Subclinical Changes in Lung Physiology | Symptoms | Reference |
|---|---|---|---|---|---|---|
| 0.25 | 2 hr | Hyperreactives[a] | Yes | Not statistically significant | No | 19 |
| 0.25 | 4 hr | Hyperreactives[a] | No | Not statistically significant | No | 20 |
| 0.37 | 2 hr | Normals[b] | Yes | Not statistically significant | No | 19 |
|  |  | Hyperreactives[a] | Yes | Statistically significant[d] | Yes[e] |  |
|  | 4 hr | Hyperreactives[a] | No | Statistically significant[d] | Yes[e] | 20 |
| 0.20-0.53 | 3-4 consecutive days | Individuals with emphysema | No | Statistically significant[d] | Yes[e] | 26 |
| 0.50 | 2 hr | Normals[b] | Yes | Statistically significant[d] | Yes[e] | 19 |
|  | 4 hr | Normals[b] | No | Not statistically significant | No | 20 |
|  |  | Hyperreactives[a] | No | Statistically significant[d] | Yes[e] |  |
| 0.60-0.8 | 2 hr | Normals[c] | Yes & No | Statistically significant[d] (with or without exercise) | Yes[e] | 8, 38 |
| 0.90 | 5 min | Normals[c] | Yes | Statistically significant[d] | Yes[e] | 23 |

[a] Individuals with history of cough, chest discomfort, or wheezing associated with allergy or exposure to air pollution.

[b] Individuals without a history of cough, chest discomfort, or wheezing associated with allergy or air pollution exposure.

[c] Individuals considered healthy on the basis of medical history and physical status and with no respiratory disease.

[d] Statistically significant changes occurred in expiratory flow rates, total lung capacity, diffusing capacity, or pulmonary resistance.

[e] Symptoms include cough, wheezing and chest discomfort.

in the blood. The points at which inhibition occurs in this pathway are seen in Figure 2. Because of the inhibition of enzymes, metabolites accumulate in the blood and eventually appear in the urine. The increased free erythrocyte protoporphyrin (FEP), erythrocyte zinc protoporphyrin (ZPP), free erythrocyte coproporphyrin, increased urinary coproporphyrin, and increased δ-aminolevulinic acid (ALA) in serum and urine indicate that these are the steps in heme biosynthesis most susceptible to inhibition by lead in man. Lead in blood and urine and changes in the levels of the above metabolites and δ-aminolevulinic acid dehydratase (ALA-D) have been used to estimate response to lead exposure. Two of these indicators, ALA-D and protoporphyrin in blood, have been selected for discussion since they are sensitive indicators of lead absorption.

Until recently blood lead was the most widely used biological indicator of lead exposure. Four ranges of blood lead concentrations have traditionally been considered to indicate the following conditions (12):

1. "Normal": range 5-40 μg/100 ml
2. Undue absorption: > 40 μg/100 ml
3. Increased lead absorption: 50-80 μg/100 ml
4. Clearly evident clinical lead poisoning:
   usually associated with concentrations > 80 μg/100 ml.

However, recent evidence suggests that blood lead is not the best available indicator of exposure to lead but that inhibition of ALA-D is the most sensitive biological manifestation of lead exposure. There is a negative relationship (Fig. 3) between blood lead and ALA-D, particularly in the range of 15-70 μg lead/100 ml blood (35,37,39). Some studies suggest that there is a no effect level at 10-15 μg lead/100 ml (17,39), while others indicate that there is no blood lead level below which no inhibition occurs (22,31).

The biological significance of ALA-D inhibition is uncertain. However, since ALA-D is not only required for hemoglobin synthesis but also for the formation of a number of heme containing respiratory

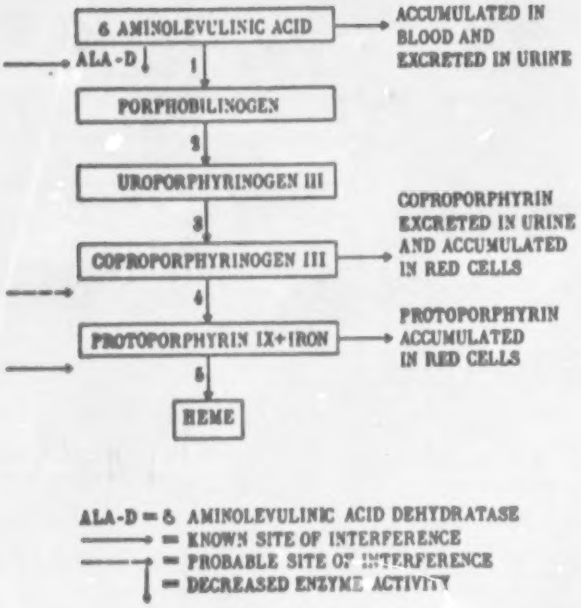
δ AMINOLEVULINIC ACID
ACCUMULATED IN BLOOD AND EXCRETED IN URINE
ALA-D
1
PORPHOBILINOGEN
2
UROPORPHYRINOGEN III
3
COPROPORPHYRINOGEN III
COPROPORPHYRIN EXCRETED IN URINE AND ACCUMULATED IN RED CELLS
4
PROTOPORPHYRIN IX+IRON
PROTOPORPHYRIN ACCUMULATED IN RED CELLS
5
HEME
ALA-D = δ AMINOLEVULINIC ACID DEHYDRATASE
= KNOWN SITE OF INTERFERENCE
= PROBABLE SITE OF INTERFERENCE
= DECREASED ENZYME ACTIVITY

Figure 2. Biosynthesis of heme (13).

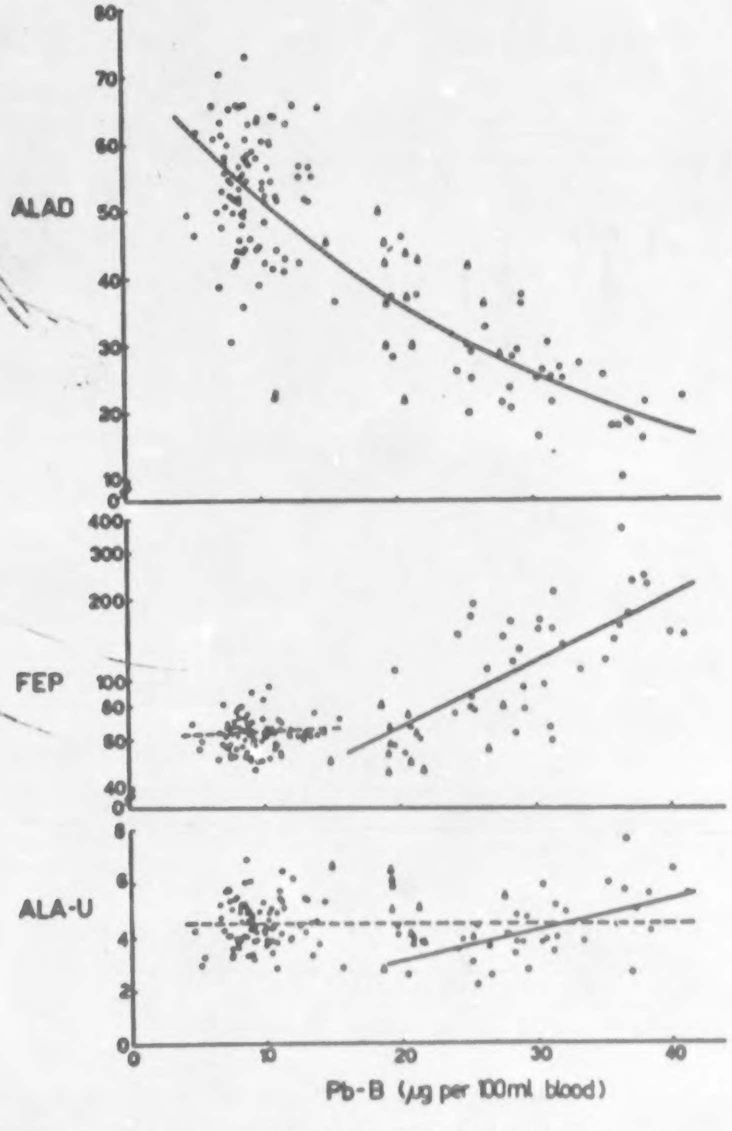

Fig. 3 Relationships between lead concentration in blood (Pb-B) and three biological measurements in children: ALAD (δ-aminolevulinic acid dehydratase activity expressed in nmol δ-aminolevulinic acid transformed/min/ml erythrocytes); FEP (free erythrocyte porphyrin expressed in μg/100 ml erythrocytes): and ALAU (δ-aminolevulinic acid in urine expressed in mg/g creatinine). *Open circles,* rural children; *solid circles,* children living less than 1 km from lead smelter; *triangles,* children living 2.5 km from lead smelter. *Dotted line:* FEP, regression line for rural children (N = 92); ALAU, regression line for total child population (N = 143). *Solid Line:* ALAD, regression line for total child population (N = 143); FEP, regression line for children living in lead smelter area (N = 51); ALAU, regression line for children living less than 1 km from lead smelter (N = 37). (31)

enzymes, and since it is present in a number of tissues (21), it is possible that ALA-D inhibition in these tissues does have health significance. It has been found that ALA-D is inhibited in brain, liver, kidney, and bone marrow of lead poisoned animals (16), and in brains of suckling rats of lactating rats fed lead (25). This is a possible implication for development of mental retardation in lead poisoned children.

Increased lead burden interferes with iron utilization and results in the accumulation of protoporphyrin in erythrocytes, excess iron in the blood, and ultimately anemia. The elevation may persist for months, even after blood lead has decreased considerably (39). Protoporphyrins are measured as FEP or ZPP. There is a positive correlation between the log FEP and blood lead (Fig. 3). Threshold values for the FEP response have been observed. One study revealed a threshold at approximately 20 µg/100 ml blood (31). Data suggest that FEP increases more rapidly in adult females and children and that the increase occurs sooner in children (31). The no response level (defined as that blood lead level which does not induce a specified response in > 5% of subjects) occurs at a blood lead level of 20-25 µg/100 ml in adult females and children, and 25-30 µg/100 ml in adult males (39). Again the health significance of this physiological change is not clear. Some investigators (31) suggest that an increased FEP level is not tolerable, since accumulation of free protoporphyrins might exert a toxic action itself (29) not necessarily related to the heme production.

A further illustration of subclinical effects is provided by evidence of peripheral nerve damage in workers. A study of lead exposure and the peripheral nervous system investigated effects in workers with no clinical symptoms whose blood lead never exceeded 70 µg/100 ml (32). The main findings were slowing of conduction velocities of the median and ulnar nerves (related to inner and larger bone of the forearm).

Electromyelographical abnormalities were also observed. The well-being of these workers did not seem to be influenced by the neuropathy; however, the investigators regarded subclinical neuropathy as a more serious effect than the quite reversible alterations in heme synthesis, because the nervous system has a poor regenerative capacity. Similar findings have been noted in children. A study of children, all apparently healthy, living near a primary lead smelter in Idaho revealed a significant decrease in motor nerve conduction velocity in 5-9 year olds. Their blood lead levels were $\geq$ 40 µg/100 ml as compared with matched controls with levels < 40 µg/100 ml (6).

Subclinical effects are of greater concern to hypersusceptible groups. Young children are more susceptible than adults because of their immature physiologic development, especially the central nervous system (36). Because of their tendency to place non-food substances in their mouths, children are more likely to be exposed to sources of lead, such as lead in paint, soil and dust. Furthermore, studies suggest that the rate of intestinal absorption of lead is greater in children than adults (1). Dietary deficiencies of calcium, copper and iron have been found to increase absorption of lead from the intestinal tract of rats (28). Therefore, it is possible that increased absorption occurs in humans with dietary deficiencies. As mentioned previously, females and children experience a more rapid increase in FEP than males. Pregnant women are at high risk to lead exposure especially in industrial situations, and fetal development may be affected (9).

Certain medical conditions may increase susceptibility to the effect of lead. It has been suggested that the excessive use of alcohol may modify a person's susceptibility (24,27). Certain blood disorders such as anemia or glucose-6-phosphate dehydrogenase deficiency may increase susceptibility (27). Lead exposure in animals results in increased susceptibility to infection (15) and thus may have a similar effect in man.

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# CHARACTERIZATION OF ENVIRONMENTAL HAZARDS: IDENTIFICATION AND QUANTITATION OF STRESSORS

R. A. Wadden

Incorporation of health considerations into project planning begins with identification of the potential health hazards which may be discharged. Evaluation can be roughly divided into three major areas: what goes into the process, what the process is supposed to do, and how the process is operated. As an example, the basic physical-chemical structure of the coal fed to fossil-fueled power plants will result in greater amounts of carcinogenic polycyclic hydrocarbons in the stack gas, compared with the amounts from burning an equivalent quantity of natural gas (7) (Fig. 1).

What the process is supposed to do and how it is operated may be two quite different considerations. When operating continuously under steady conditions, the carbon monoxide emissions from an auto exhaust pipe are typically only 10-20% of the emissions from the same engine initially starting up (9). The same is true of many chemical and energy processes where startup emissions, due to nonsteady-state operating conditions of temperature, pressure, and flow, are much in excess of those during design operation. Sulfuric acid plants require certain process components to operate at temperatures up to 1150°F. When starting up such plants, 6-24 hours may be required to heat all the equipment to design temperatures. Unless proper control measures are instituted, the discharge of sulfuric acid mist and $SO_2$ will be much greater than under steady state conditions. Finally, the most sophisticated control device in existence still depends on a man to turn it on (or off), and maintain it.

After identification of hazardous materials, the amounts and potential environmental release rates must be quantitated. Information is available for pollutants like particulates and nitrogen oxides for such

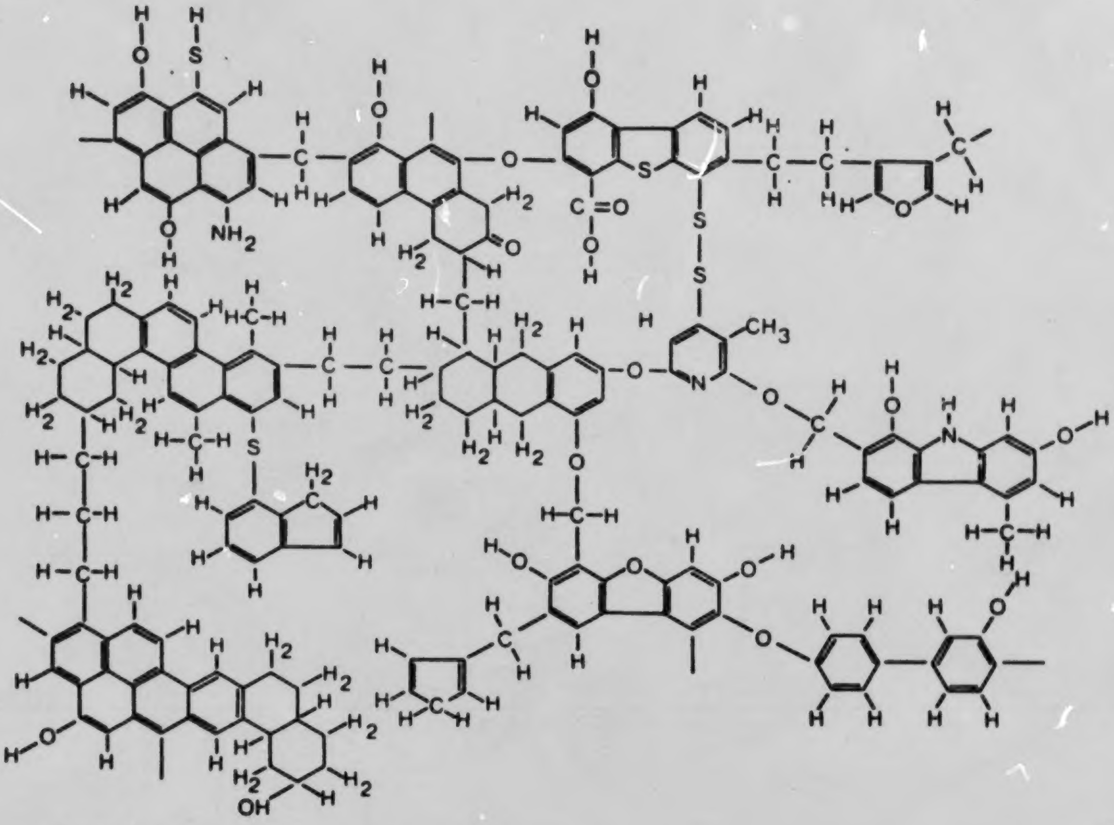

FIGURE 1. A representative bituminous coal structure.

things as new automobiles and oil fired power plants (9). Table 1 gives a set of emission factors which can be used to estimate emissions from such power plants. The data is harder to come by for: (a) less commonly considered pollutants, such as benzo(a)pyrene from coal combustion; (b) new, untested technologies, such as coal gasification; and (c) older, existing pollution sources, such as diesel buses and trucks, which have been poorly maintained. Amounts of a particular pollutant discharged to air, water and soil must be measured or estimated. Table 2 shows some estimates for trace metals from a coal gasification process (10).

Often overlooked in project evaluation, are the baseline conditions of the environment. Before process installation or use in an area, determination of existing levels of air, water and soil contamination must be determined for those hazardous materials which may be discharged. For pollutants which have short-term physiological effects, such as ozone for one hour or CO for one and eight hours (8), it is particularly important to obtain data for the worst short term interval during the year. As an example, municipal parking garages adjacent to shopping districts are often designed on the basis of the 30th busiest commercial sales day of the year. Exposure of individuals with coronary heart disease to carbon monoxide at levels from 37-63 ppm for 1.5 to 4 hours has resulted in statistically significant increases in anginal distress (2-4). The carbon monoxide in air standards designed to protect these and other hypersusceptible individuals call for limitations of 35 ppm over one hour, and 9 ppm over eight hours, to be exceeded no more than once per year (8). Hence, use of the process design criteria for health considerations is inadequate. Baseline information needs to be gathered for the highest and second highest hourly and eight-hourly average for CO, before the impact of a new shopping complex can be evaluated.

Since air and water pollutants, and to a lesser extent soil contaminants, do not entirely remain where they are initially discharged,

Table 1. Emission Factors For Fuel Oil Combustion.*

| | Type of Unit: Industrial and Commercial | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Power plant | | Residual | | Distillate | | Domestic | |
| Pollutant | lb/$10^3$gal | kg/$10^3$ liters | lb/$10^3$gal | kg/$10^3$ liters | lb/$10^3$gal | kg/$10^3$ liters | lb/$10^3$gal | kg/$10^3$ liters |
| Particulate | 8 | 1 | 23 | 2.75 | 15 | 1.8 | 10 | 1.2 |
| Sulfur dioxide† | 157S | 19S | 157S | 19S | 142S | 17S | 142S | 17S |
| Sulfur trioxide† | 2S | 0.25S | 2S | 0.25S | 2S | 0.25S | 2S | 0.25S |
| Carbon monoxide | 3 | 0.4 | 4 | 0.5 | 4 | 0.5 | 5 | 0.6 |
| Hydrocarbons | 2 | 0.25 | 3 | 0.35 | 3 | 0.35 | 3 | 0.35 |
| Nitrogen oxides ($NO_2$) | 105 | 12.6 | (40-80) | (4.8-9.6) | (40-80) | (4.8-9.6) | 12 | 1.5 |
| Aldehydes (HCHO) | 1 | 0.12 | 1 | 0.12 | 2 | 0.25 | 2 | 0.25 |

*From: Compilation of Air Pollutant Emission Factors (2nd Ed.). U.S. EPA, AP-42, 1972.

†S = % by weight of sulfur in the oil.

Table 2. Some trace elements in coal and coal gasifier gas.*

| | Concentration, lb of trace element/1000 tons of coal | |
|---|---|---|
| Element | Coal | Coal gasifier gas |
| As | 28.0 | 27.7 |
| B | 204 | 97.3 |
| Be | 3.2 | 0.1 |
| Cd | 5.0 | |
| Co | 19.1 | |
| Cr | 27.5 | |
| Cu | 30.3 | |
| F | 122 | 121 |
| Hg | 0.4 | 0.4 |
| Mn | 98.8 | 6.8 |
| Mo | 15.1 | 13.7 |
| Ni | 42.1 | |
| Pb | 69.6 | 25.2 |
| Sb | 2.5 | 1.9 |
| Se | 4.2 | |
| Sn | 9.6 | |
| V | 65.4 | |
| Zr | 145 | |

*From: Wadden, R. A. Coal hydrogenation and environmental health. Environ. Health Perspect. 14:201-206, 1976.

the dispersion of toxic materials must be estimated. The effects of topography and meteorology are of importance in this determination for air, and those of hydrology for water. Transport of materials in air and water to locations remote from their emissions is well documented. As examples, industrial particulate plumes have been traced for tens of kilometers from their sources (6). Elevated sulfate concentrations, measured in rural and suburban air, have been attributed to sulfur dioxide sources even more distant (1,5). Not as obvious, but of paramount importance, is the level of degradation produced, in the immediate area and remotely, in air, water and soil by source emissions. Transport of hazardous materials may be physical, as in the case of automobiles and airplanes, as well as meteorological.

The evaluation of a specific energy-producing or using facility should then include consideration of most of the following categories:

1) Raw materials - chemical and/or physical structure constituents; amounts.
2) Process design - conversion conditions; product types and amounts; "trace substances"; solvents; absorbents; scrubbing liquids; catalysts; other materials in the process; material balances on major and "trace" components.
3) Process operation - startup; shutdown; pollutant discharges during expected intermittent operation.
4) Emissions - pollution control equipment; maintenance of control devices; transfer of pollutant from one phase to another; production of undesirable byproducts.
5) Interaction of site with source - meteorology; topography; hydrology; physical design of installation; transportation.
6) Interaction of source with existing sources - regional air and water considerations; existing air and water quality; non-degradation; air and water quality modeling.

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# CHARACTERIZATION OF ENVIRONMENTAL HAZARDS:

## DISPERSAL OF POLLUTANTS IN AIR*

L. R. Babcock, Jr.

The transport of air pollutants from sources to receptors should be a vital consideration for the planner. Emissions might be considered of little consequence until they do reach and cause damage to human or other receptors. How do these waste products or emissions get from the sources to receptors? They travel through the air, but the mechanisms of transport are complex. We know that all of the emissions from a source do not drop on a single receptor; rather, they spread and are diluted as they travel downwind from a source.

Under some conditions the emission will be greatly diluted in a short time. At other times the emission will not be diluted very much at all. If we are to estimate damages, we need to know how much of the emissions from a given source will get to a given receptor point.

Our knowledge of urban meteorology remains incomplete, but this doesn't mean we should ignore dispersion calculations. We are interested in spatial patterns, in relationships between sources and receptors. We want to know how the arrangement of sources affects the air quality at receptor locations. Various kinds of atmospheric pollution dispersion models can be employed. Figure 1 makes the point well. The dispersion model should relate the sources, meteorology, and spatial patterns to air quality at receptor points.

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*Adapted from "Air Pollution Dispersion Modeling: Application and Uncertainty" by R. J. Allen, L. R. Babcock and N. L. Nagda Regional Science Perspectives 5,1-26, 1975.

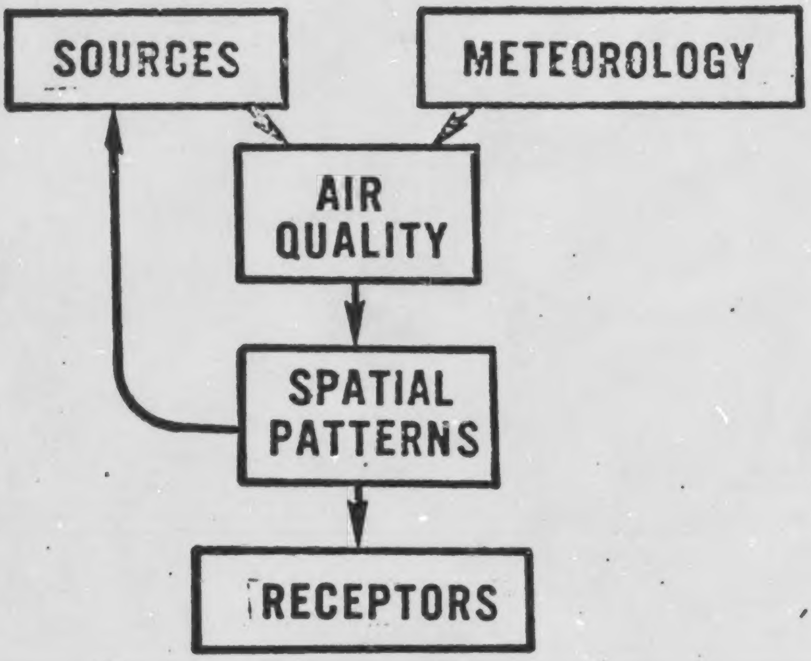


Figure 1: Schematic for an air quality system.

A dispersion model should give us a means for determining where to pu the next smoke stack so that the best possible air quality will result. What kinds of models are there? What information is needed in order to apply a model? How well do the models work? These are some of the questions I will try to address.

Right at the beginning, let me admit that, in general, the present state of the art does not allow for a very accurate prediction of air quality at some specific point in time and space. There are many reasons for this, and some of them will be pointed out as we go along.

## TYPES OF DISPERSION MODELS

In general there are three basic types of dispersion models: box models, plume models and puff models. The box model is the simplest and the puff the most complicated.

Each model requires a coordinate system so that we can locate things within the urban region. The most common coordinate system (Fig. 2) used for real cities is cartesian. The x-axis and the y-axis are on the ground and the z-axis is vertical. So any point P, whether a source or a receptor, can be located in terms of coordinates x, y, and z.

Now let's look specifically at each model. The box model (Fig. 3) is the simplest. It is called a box model for obvious reasons. The region is approximated as having definite sides and a lid as well as a flat bottom at ground level. The sources within the box are modeled as a completely-mixed and dispersed area source.

The equation (Fig. 3 ) is shown here. The concentration (C) anywhere in the box is simply a function of the incoming pollution ($q_o$), the total source strength within the box (Q), the crossectional area of

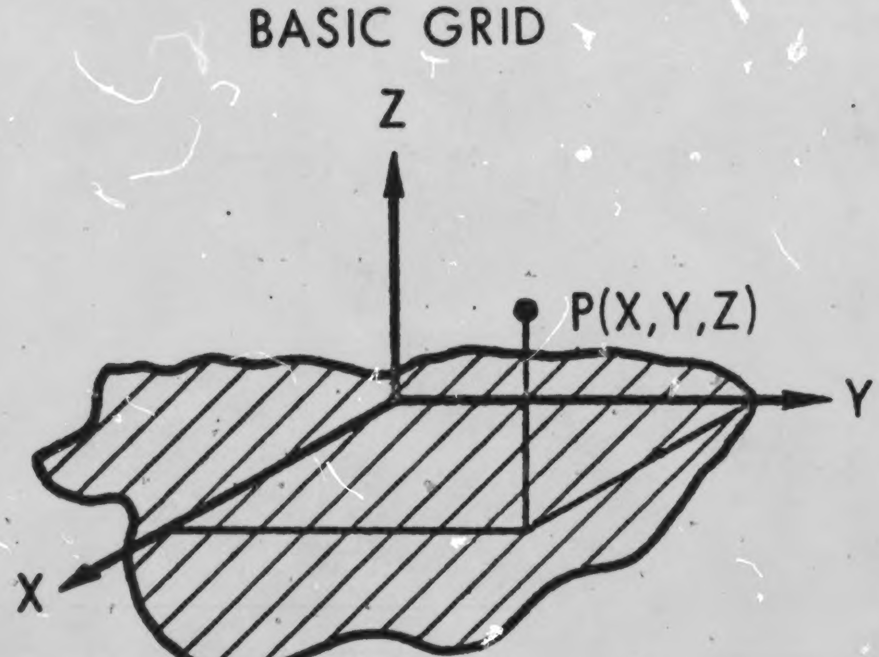
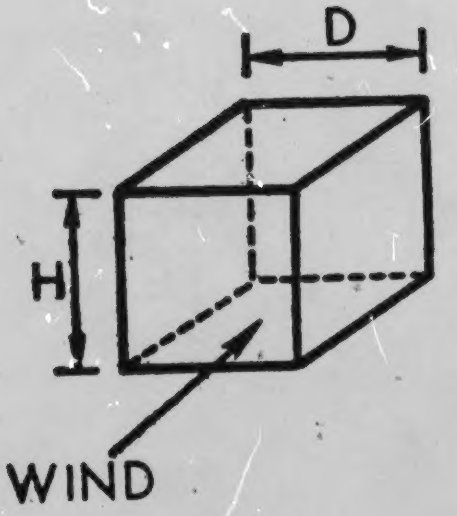


Figure 2: Cartesian coordinates.



$$C=q_o + \frac{Q}{D \cdot H \cdot U}$$

Figure 3. Box model.

an end of the box (D times H), and the wind speed (U).

Note that the city's length has no effect on the result; the important dimensions are simply the crosswind distance and the mixing height.

The calculations for the box model are straight-forward and useful for those back-of-the-envelope approximations which can help define a problem. The limitations are obvious. Urban emissions from point and line sources do not get uniformly back mixed within a clearly defined rectangular volume. At certain places within the box, the pollution level would be much higher or much lower than that calculated.

Plume models have been developed as a more realistic representation of pollutant dispersion. With realism, unfortunately, comes complexity. With a plume model it is possible to treat sources individually rather than combining them as in the box model(10,12). As a plume moves downwind (Fig.4) it spreads vertically and horizontally. The concentration varies in space. Plume equations, such as the gaussian one shown in Figure 5, enable us to individually characterize each source. The total concentration at a receptor point is the sum of the contributions from all sources plus the background concentration.

Here, for a point source, the concentration (C) is a function of the source emission rate (Q), mean wind speed (U), vertical spread of the plume (sigma z), lateral spread of the plume (sigma y), effective stack height (H) and the coordinate positions of the source and the receptor in terms of x, y and z. The equation is obviously much more complicated than that for the box model, but it readily can be solved. Don't worry about those mysterious sigma terms; these have to do with the stability of the atmosphere. You pick them out of tables or graphs. Not very satisfying, but more about that later.

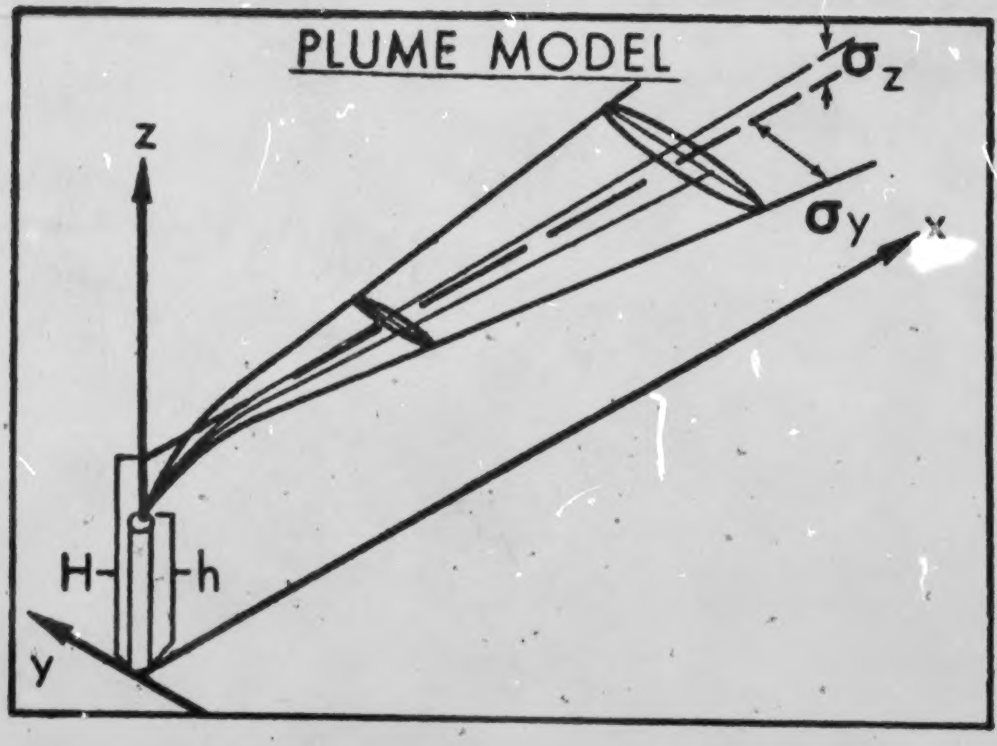


Figure 4. Plume dispersion.

# PLUME EQUATION

$$C = \frac{Q}{2\pi \sigma_y \sigma_z u} \exp\left[-\frac{1}{2}\left(\frac{Y}{\sigma_y}\right)^2\right] \cdot \left\{ \exp\left[-\frac{1}{2}\left(\frac{Z-H}{\sigma_z}\right)^2\right] + \exp\left[-\frac{1}{2}\left(\frac{Z+H}{\sigma_z}\right)^2\right] \right\}$$

**Figure 5.** Gaussian plume equation.

Also note that the height term, H, is rarely the height of the stack, since plumes are usually bouyant and tend to rise considerably above the stack exit.

Despite the complexity, this equation still does not realistically characterize all kinds of plume dispersion. Many assumptions remain. The ground surface is still assumed to be smooth and flat rather than covered with buildings of various heights. The plume is assumed to be in steady state. That is, its size, shape, and orientation do not change over time. The emission level is assumed to be constant. Note that there are no time variables in the equation.

In summary, these equations were developed for use with single sources in uncomplicated rural areas over relatively short travel distances such as one to two miles. The risks are obvious when such equations are applied to large, complex, multiple-source, urban regions where dispersion or source-receptor distances can exceed thirty miles. The equation doesn't always work in the country either. Because of tricky meteorology, plumes emitted at different altitudes can go in completely different directions, even curve in the process. Problems are to be expected, and I will discuss some of them later when we compare the performance of some of the dispersion models.

The final basic model type which I want to mention is the puff model (9,10). Here the emissions are treated as individual puffs (Fig.6). If the weather chages, a simulated downwind puff can be made to act accordingly. Here is a model which includes time variables. Otherwise, application of the puff model is similar to that for the plume model.

In addition to the variables used in the plume equation, this model (Fig. 7) utilizes a fundamental time increment (delta), and two additional time terms (t and t') which characterize release and reception of the puff.

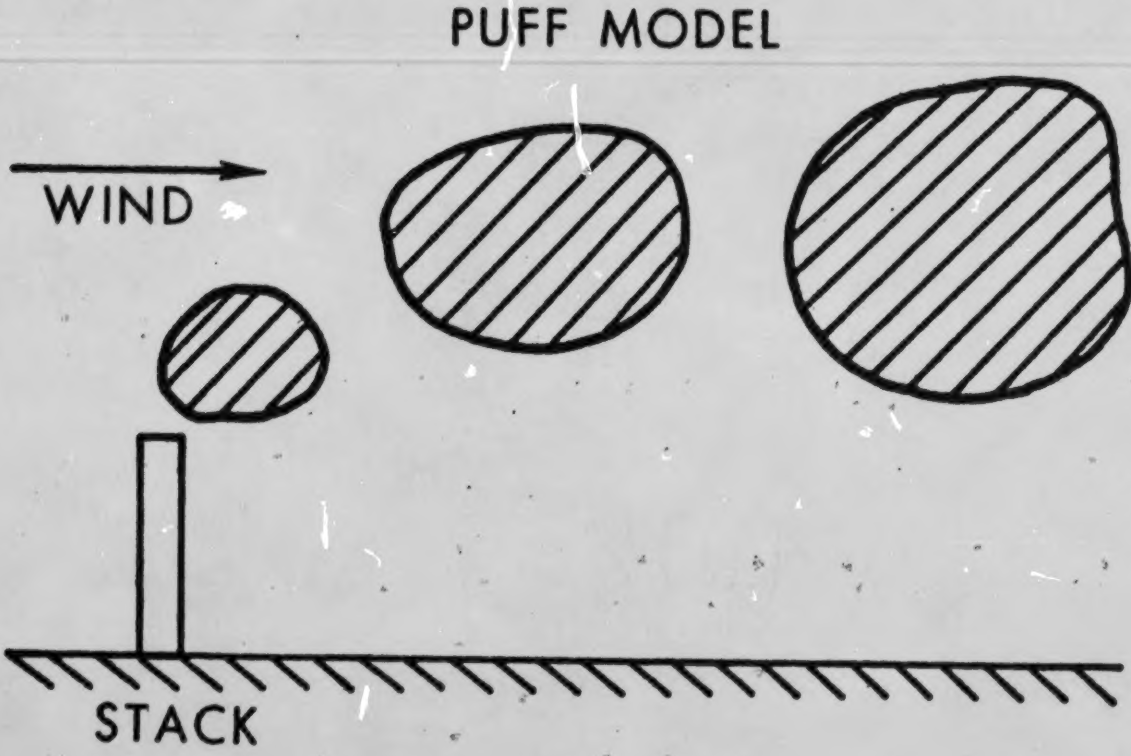


Figure 6. Puff model.

$$C = \sum_{n=1}^{m} Q_{m-n+1} \int_{(n-1)\Delta}^{n\Delta} dT$$

$$\cdot \left\{ \frac{\exp - \left[ \frac{(x-UT)^2}{2\sigma_x^2} + \frac{y^2}{2\sigma_y^2} + \frac{z^2}{2\sigma_z^2} \right]}{(2\pi)^{1.5} \; \sigma_x(T)\, \sigma_y(T)\, \sigma_z(T)} \right\}$$

$$\text{where} \quad T = t - t'$$

Figure 7. Puff model equation.

The integration and the time considerations make the equation difficult and expensive to apply to a complex multiple-source region, unless large core, low-cost computer facilities are available.

These three basic model types (box, plume and puff) form the basis of almost all dispersion simulations which are in use today.

## METEOROLOGICAL AND DISPERSION VARIABLES

Before we look at how well the models perform, let's look in a little more detail at the model variables. The results obtained from any model, unfortunately, are completely dependent upon the quality or reality of the input data. Problems with input data are in part responsible for the less than satisfying showing of any model.

Perhaps an estimate of what is being emitted, and where, is most important. Of course, it is unlikely that we would know exactly what is coming out of every stack, in what quantity, at every instant. Instead, we settle for crude estimates of emission rates (7). Emission estimates are usually based upon emission factors and production data. This approach, of course, masks all significant, short-term variations in emissions.

Thus dispersion calculations often cannot realistically characterize short-term pollution situations. This is one reason why I suggest that improvement of ambient air quality be measured on an annual basis. Where necessary, annual averages can be related statistically to short-term situations, but that's a topic best left for another paper.

Emission data for whatever time interval must be coupled with information about the atmosphere. It would seem that it would be easy to get data on wind speed and direction. In most urban areas, weather data are collected at the airport which is often out in the country, several miles from the city. We know that wind speed and direction can change as air moves from flat farm land into hotter, built-up areas of a city. To make matters worse, wind speed and direction change with altitude (11,12), another reason why single-altitude airport values cannot adequately characterize the condition of the air mass over a city. Unfortunately, multiple-location weather data are not routinely collected in most cities. Airport data are used because they are usually the only data available.

Next, let's return to those mysterious sigmas in the plume and puff equations (12). These characterize the extent of plume dispersion in the vertical and in the horizontal-crosswind directions. These values also are influenced by atmospheric conditions, but in ways which are much more difficult to characterize than wind speed and direction.

Stability is a way of characterizing the atmosphere's resistance to change. If the atmosphere is unstable, the sigmas are large, and the pollutants will be diluted and dispersed quickly. If the atmosphere is stable, the sigmas are small, and little dispersion takes place.

The modelers have come up with an indirect characterization of stability, one based on surface wind speed and the amount of cloud cover (Fig. 8) (12). These indirect proxies are used to define six classes of stability. First you estimate the stability class and then use a graph (Figure 9) to obtain the sigma (12). The largest source of error involves the selection of the stability class. Note the order of magnitude variations on the log - log plot.

To make matters worse, the spreading of plumes is fairly well understood only for short travel distances-- a few kilometers. For longer distances, we have little experimental information. In a large urban

| WIND (mph) | SUNSHINE | | |
|---|---|---|---|
| | HI | MED | LOW |
| <5 | A | A-B | B |
| 5-7 | A-B | B | C |
| 7-11 | B | B-C | C |
| 11-14 | C | C-D | D |
| >14 | C | D | D |

| | NIGHT CLOUD COVER | |
|---|---|---|
| | >1/2 | <1/2 |
| <5 | - | - |
| 5-7 | E | F |
| 7-11 | D | E |
| 11-14 | D | D |
| >14 | D | D |

Figure 8. Stability characteristics.

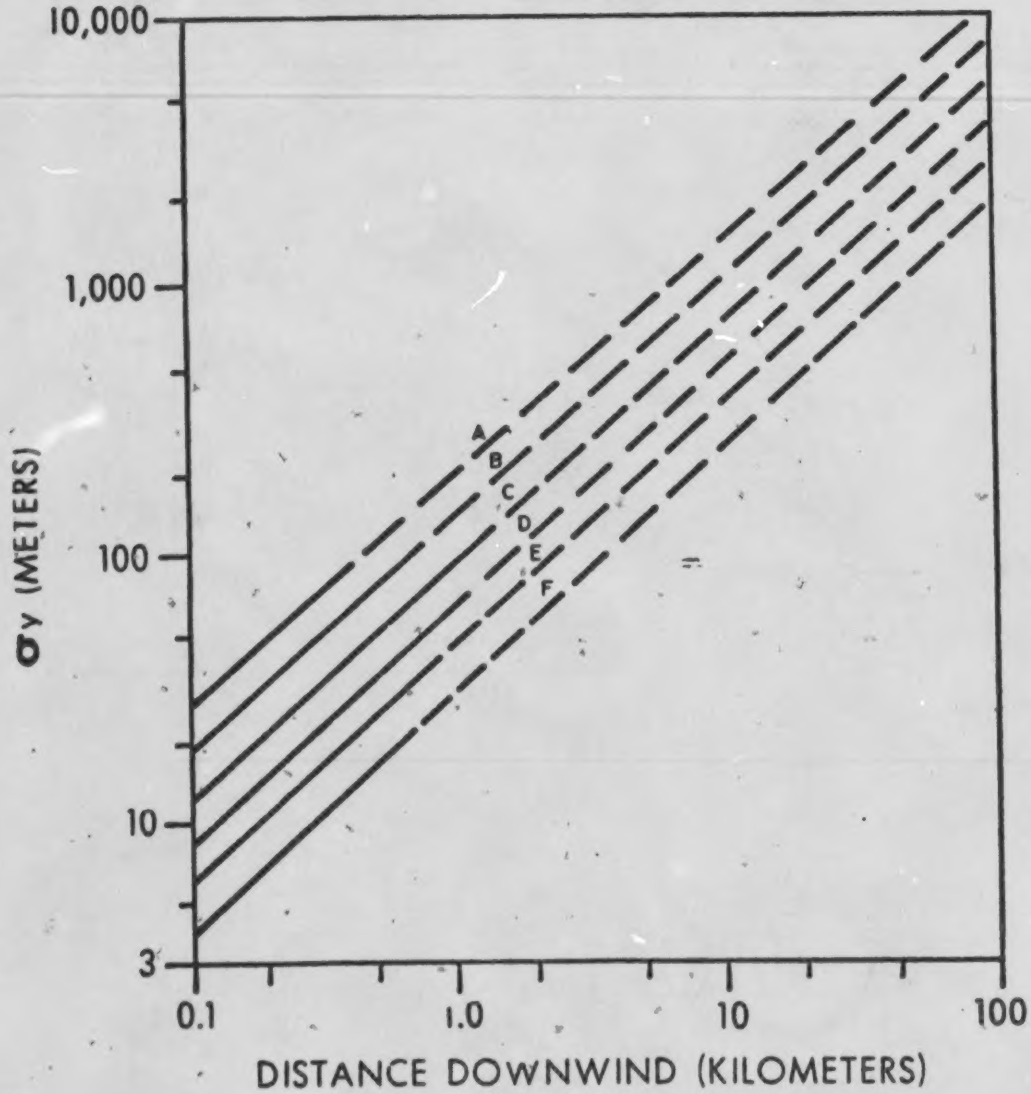


Figure 9: Sigma y-distance relationship.

area, travel distances of 20, 50, or even 100 kilometers are of interest. For most of the plume-travel distance, it is hard to say just what is happening to the plume.

Also needed in the plume equations are a value for the effective stack height of each source. Several different equations have been proposed for estimation of plume rise. The Holland equation or the Davidson-Bryant equation are examples (7). Both equations are functions of stack and atmospheric variables. However, the two equations generally will not give the same answer under the same conditions.

Mixing depth is another important variable. It is the height of the invisible lid in the box model. It didn't appear in the plume or puff equations, but it defines the vertical limit of the three-dimensional air space in which the equations can be applied. The mixing depth or mixing height (these terms mean the same thing) is the height to which the pollutants can be expected to readily mix in the atmosphere. When temperature inversions exist, the mixing height can be clearly defined. Usually, the situation is less certain.

Mixing height, like stability, is not routinely measured directly. In fact, for mixing height, a standard method of estimation has yet to be agreed upon (8). We know such barriers exist, but we can't be sure of the exact height or whether the barrier has holes in it. Sometimes the vertical flow of pollutants is merely slowed rather than being completely confined.

Estimates of mixing depth are based upon vertical temperature soundings as obtained from weather balloons. Locations of these soundings are usually remote from urban areas. Such measurements do not reflect urban and other geographical abnormalities such as urban-heat-island or lake effects. These phenomena can dramatically influence the mixing depth.

When mixing depth is low (a few hundred feet) (Fig. 10), the effect upon urban air quality can be profound. Inversions can effectively trap pollutants in a small volume near the ground. In unusual cases (Fig. 11), tall stacks can cause the reverse to be true, with emissions taking place above the mixing depth. I suggest that you not rely on this form of air quality management.

What happens if a plume hits the barrier? Most variants of plume and puff models assume the pollution is reflected back (Fig. 12) as shown here when it hits either the ground or the mixing lid (10,11). Eventually these constraints cause the pollutant concentrations to change from the plume configuration (Fig. 13) into that of the evenly dispersed constant concentration of the box model.

Before moving on, I want to say a little more about Chicago's lake breeze (3). Lake Michigan or other bodies can have an important effect upon air quality. Under certain conditions the lake recirculates our pollutants. Figure 14 is the trajectory of a balloon released into an onshore or lake breeze. It rose, then went opposite to the lake breeze. Then it descended and returned in the onshore breeze; it then started to repeat the cycle. This means that pollution can be blown away only to return again, sometimes several times in the same day. The dispersion equations don't account for this sort of thing. Of course, if an onshore lake breeze is infrequent, don't worry about it. In Chicago we experience such conditions on about 60 per cent of spring and summer days. This is a significant portion of the time, even on an annual basis.

Such modeling anomalies should be investigated in regions located near large bodies of water. The seemingly straightforward dispersion equations overlook the complexities of marine effects and unusual topography, as well as the aforementioned urban heat island. Any of these can significantly affect the dispersion of urban pollutants,

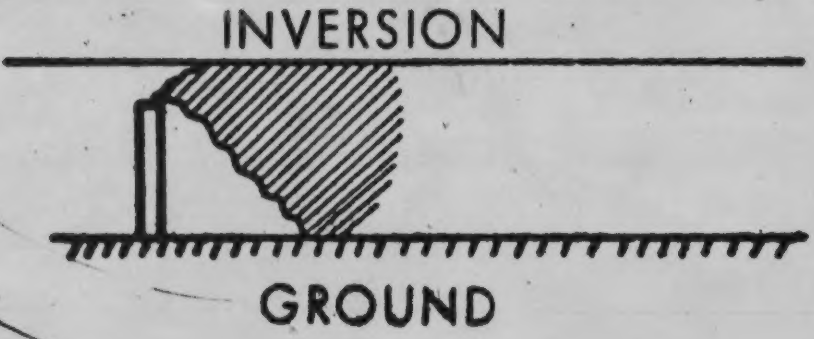


Figure 10. Emissions trapped in an inversion.

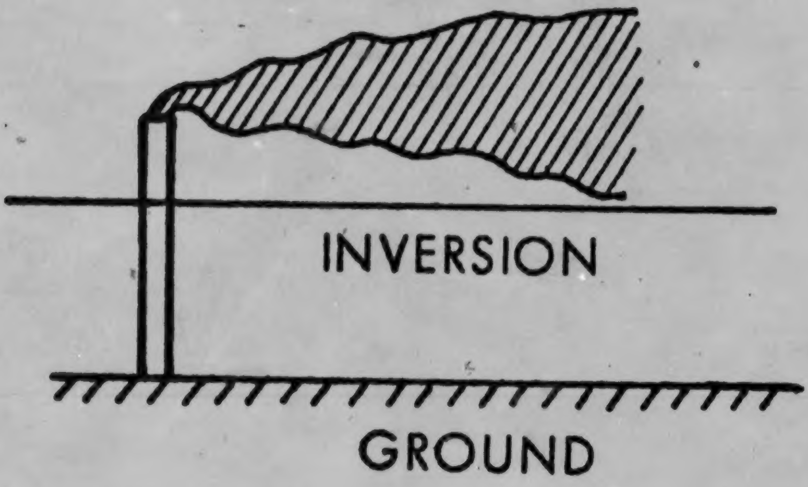


Figure 11. Emissions above inversion.

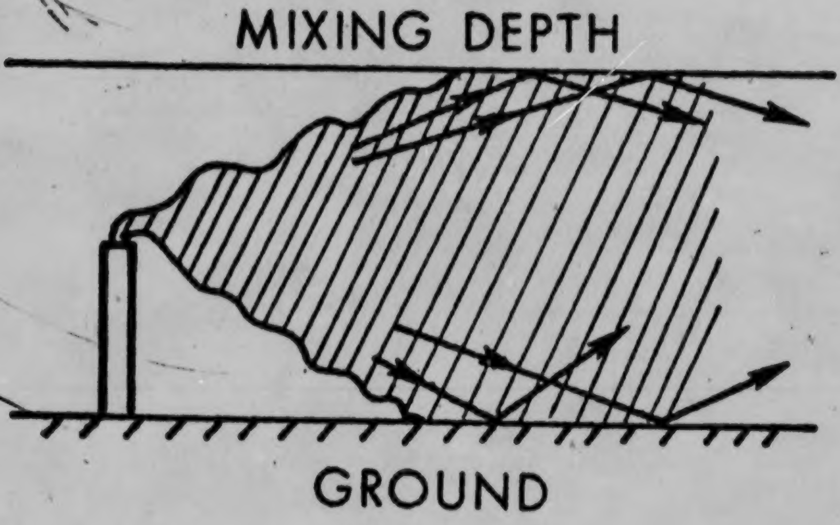


Figure 12. Plume reflection.

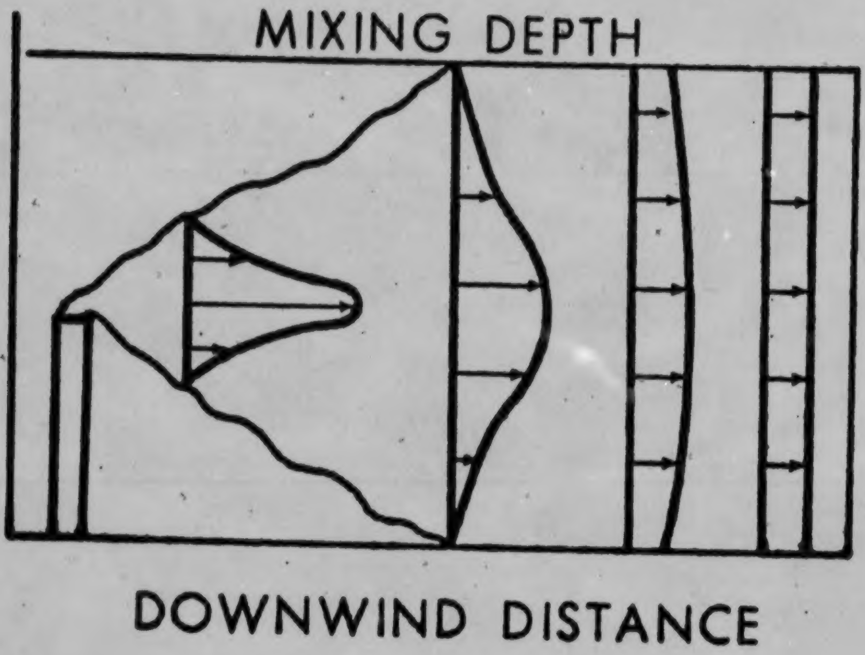


Figure 13. Constrained plume.

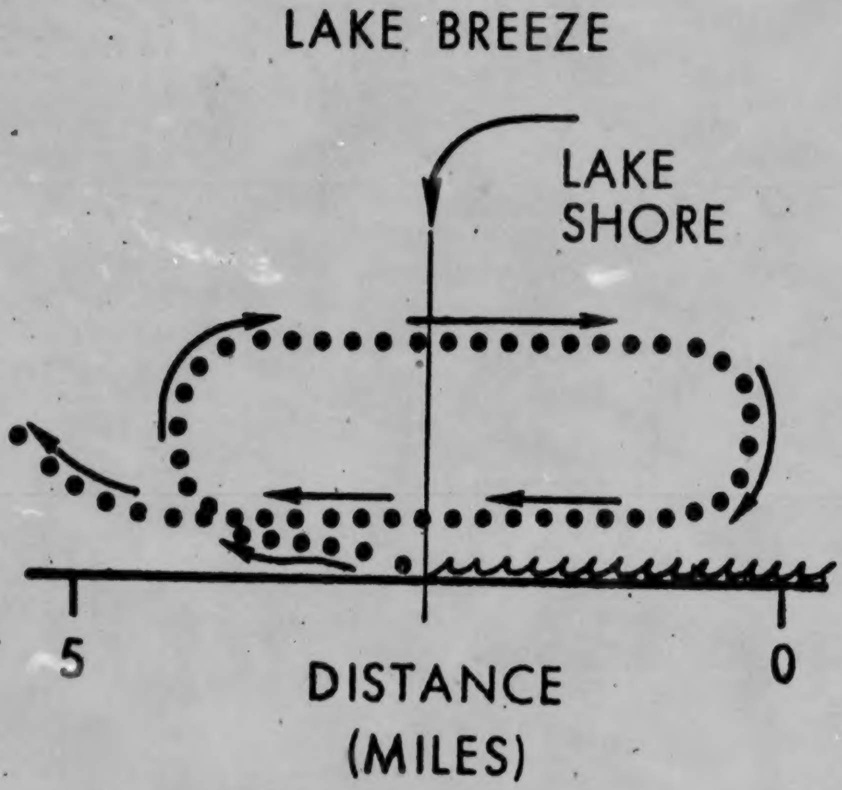


Figure 14. Pollutant trajectory in a lake breeze.

and atmospheric scientists have been notably unsuccessful in modeling these abnormalities.

In contrast, background pollution levels can be readily incorporated into plume calculations. The incoming air is not clean (Fig. 15). Incoming pollutants can be generated from either natural or from man-made sources outside the region. Incoming pollutant levels can be measured at upwind region boundaries, and these concentrations can simply be added to the results of any dispersion calculations.

The question of pollutant decay is more complicated. The pollutants don't just disperse; they do remove themselves from the atmosphere. If decay mechanisms didn't exist, we all would have been asphyxiated long ago. Pollutants are not inert. We just don't have enough information in regional air quality dispersion models to determine how fast, and how much decay occurs within the region. If the area being modeled is small and wind speeds are high, the pollutants may be quickly blown out of town without having time to react or decay. If, on the other hand, the area is large and wind speeds low, significant removal, decay, or alteration could occur.

Photochemistry is an obvious example. Oxidant is merely an unfortunate intermediate along the generally beneficial decay path by which nature attempts to rid the atmosphere of nitrogen oxides and reactive hydrocarbons. Sometimes maximum oxidant concentrations are found 50 to 100 kilometers downwind of the precursor emission points. Because of the complex kinetics, modelers usually have much less success in predicting ambient levels of these pollutants than for a relatively inert pollutant, such as carbon monoxide.

Even here, oxidation or removal must take place, since no worldwide buildup of carbon monoxide is being observed, despite the evergrowing

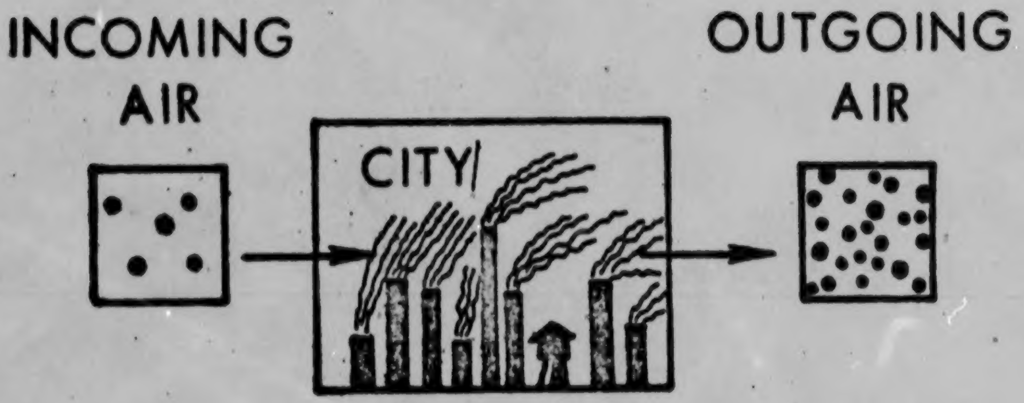


Figure 15. City emissions and air pollution levels.

number of automobiles. Again, we have no easy answers except that you recognize the risks involved in using models which exclude decay considerations.

Sometimes recirculation, pollutant decay, and background pollution are included by empirically calibrating a dispersion model. In this way a given set of input information sometimes can be forced to give the proper observed results, even when we don't understand all of the dispersion and decay mechanisms.

## MODELS: APPLICATION AND VERIFICATION

Now that you have an idea of the types of models and have been exposed to some to the problems we have in getting the numbers to plug into them, let's see how well the models work. Models have been used to simulate short term (that is, hour to hour) as well as long term (that is, year to year) conditions. I will show you some short term comparisons, even though I strongly feel that you should largely restrict the use of models to annual evaluation for multiple source systems.

Our problem is that more effort has been spent in developing models than in verifying them. When experimental verifications are attempted, it is much easier and less expensive to confine the verification monitoring to a short rather than a long time period.

This problem of monitoring is a discipline in itself. You will most likely use data generated by others. Still, you should recognize the limitations of such data. Many agencies, including the federal EPA, have discovered that they haven't always been measuring what they thought they had been measuring; sometimes the agonizing discovery is made years too late.

You probably can't afford to maintain the extensive and intensive continuous three-dimensional monitoring really needed to evaluate the long-range reliability of urban dispersion models. A single station sampling at a single height definitely cannot do the job for the entire region, no matter how carefully its sampling probes are located.

Let's get on to some past attempts at verification. Application of the box model to an urban street canyon is a straightforward example (4). The building walls clearly define the width of the box. Here are some observations (Fig. 16) from 1971 in St. Louis. Concentrations of carbon monoxide were measured at a mid-block location. Time in hours is plotted on the horizontal axis with concentration in ppm on the vertical. As you can see, there is a significant variation with time. Some of the rush hour peaks are evident. Now, using a dotted line (Fig. 17), we superimpose the results of model calculations upon the observations. The agreement is fairly good; for several of the peaks, the observed and calculated results coincide. However, large discrepancies are apparent. Here the measured concentration is over three times the calculated concentration.

What you see here is relatively good agreement, at least when compared with models in general. There are several reasons. The area being modeled is small. The geometry, that is, the dimensions of the box, is definitely defined. Single, continuous monitors for wind speed and carbon monoxide level could adequately provide the input ambient data. There was no need to estimate stability and extent of plume dispersion. Also, emissions were accurately estimated using traffic counts which were detailed enough to indicate hour-by-hour variations.

In another comparison, box models were applied to entire urban regions for yearly time periods (5). A constant pollutant concentration (C) was calculated simply by dividing the emission rate (Q) by the average windspeed (U) and the land area of the city (A): $C = \frac{Q}{UA}$.

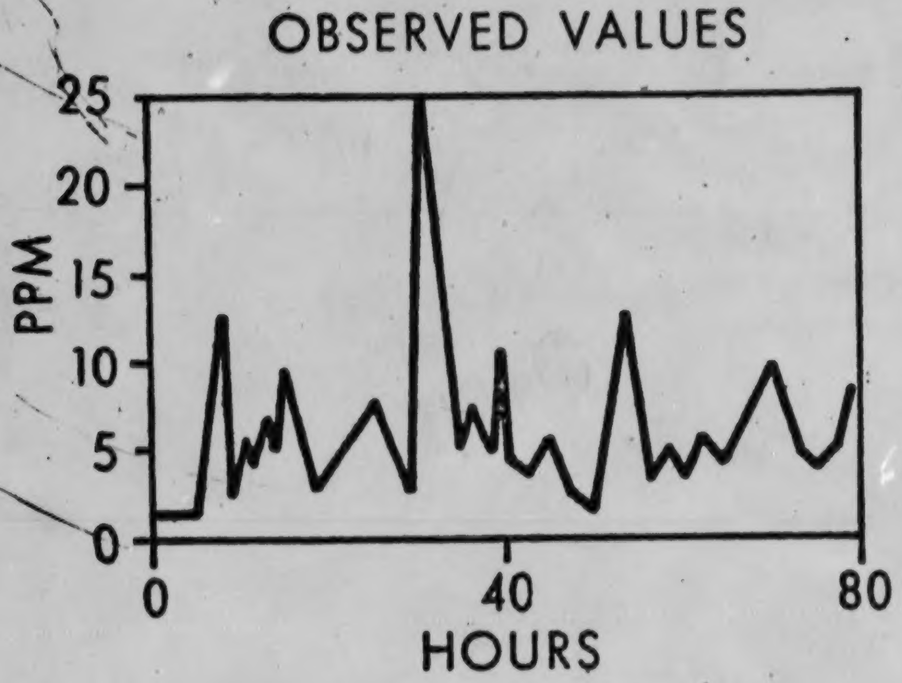


Figure 16: Carbon monoxide for an urban canyon.

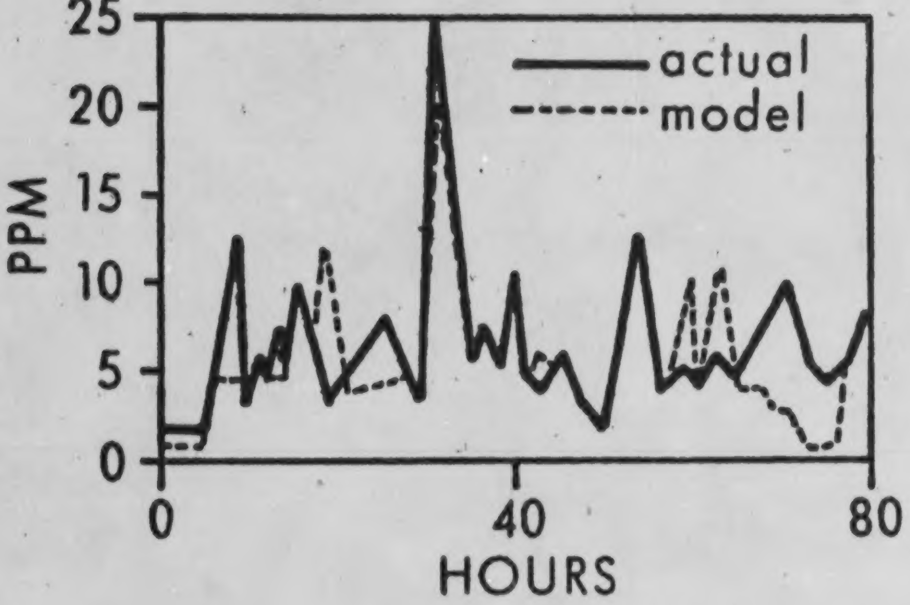


Figure 17: Comparison of box model with CO measurements.

The result is corrected by a proportionality constant (K).

Note that neither the city shape, the wind directions, nor the mixing depth are included in this extremely simple equation. Both particulates and sulfur oxides were studied for 29 U. S. A. cities in which calculated results using this equation were compared with ambient annual averages. Here are a few of the values of K (Table 1). The values for particulates are on the left, with those for sulphur dioxide on the right. For sulfur dioxide the values of K vary from 5 to 218 with an average of 50. For particulate matter, the so-called constant varies form 57 to 634 with an average of 200. These averages are for all 29 cities, not just those shown here.

The variation in K between cities is high. However, the standard error is only ± 50 per cent. Not bad for a model which almost totally ignores the urban geometry and atmospheric variables. Admittedly, this work raisesquestions. Why are the constants different for the two pollutants? How repeatable are the results? In other words, what happened the following year? Did cities keep the same constant? Did the overall averages change?

Despite the questions and the obvious limitations, this simple approach can sometimes deliver considerably useful information per man hour of effort. We should expect considerable improvement when the more sophisticated, time-consuming plume and puff models are employed.

Well, just what do we get with the plume model? Let's start with more short term results for St. Louis (10). In this case, plume equations were used to predict the total contributions of highway and airport sources upon a single monitoring or receptor location. The pollutant is carbon monoxide (Fig.18), and about three days of observations in 1964 are shown with concentration on the vertical and time on the horizontal axis. Again, some of the daily rush hours are quite

Table 1. Values of K for the Box Model*

| | K Value | |
|---|---|---|
| | Particle | $SO_2$ |
| Chicago | 154 | 81 |
| Los Angeles | 205 | -- |
| Philadelphia | 634 | 218 |
| Pittsburgh | 426 | 117 |
| St. Louis | 122 | 27 |
| Detroit | 155 | 5 |
| Cleveland | 57 | 16 |
| Average | 200 | 50 |

---

*$C = K\left(\frac{Q}{UA}\right)$

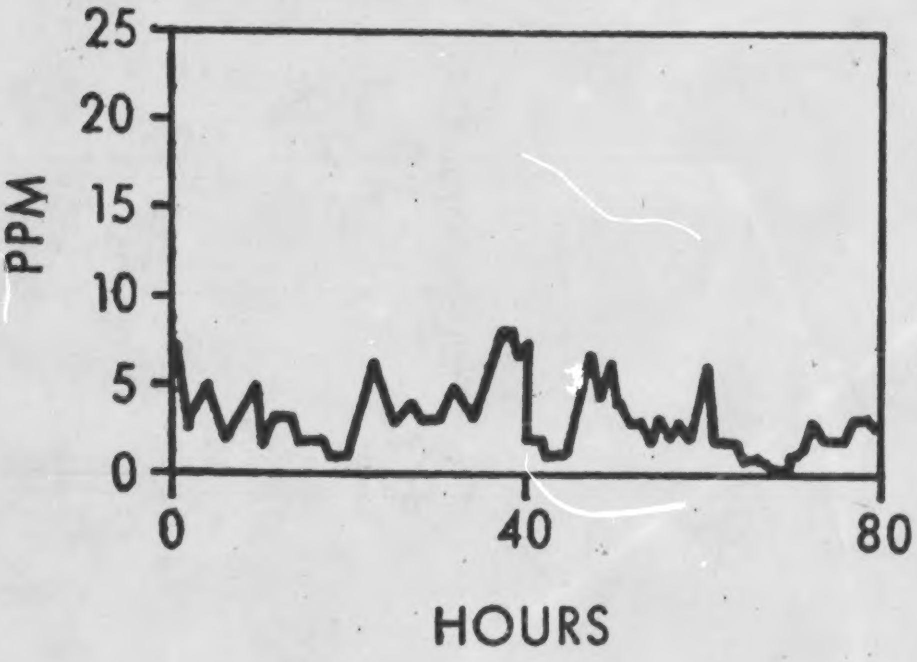


Figure 18: Measured concentration versus time for CO.

apparent. Next, we superimpose the calculated results (Fig. 19) using plume equations. Obviously, the model does not duplicate the observed conditions. For a few shorter periods of time, the model and observations are close.

At other times the model significantly deviates from observations. Here the calculated result is three times the observed measurement. Large errors also occur at these points. The errors are larger than those for the box model, but the source-receptor geometry being considered here is much more complex than in the straight-forward urban canyon example.

What about long term results? First, I must describe in greater detail the most important long-term application of plume models. In urban situations, plume calculations are rarely used one at a time. Rather, there are many sources and many receptors as well as continual variations in atmospheric conditions. The Air Quality Display Model, or AQDM for short, is a computer simulation package by which present or future air quality can be estimated (1). Within AQDM the various meteorological parameters are either entered or internally estimated: they are weighted and combined such that the model yields annual ambient air quality estimates at large numbers of preselected locations. AQDM is one of two methods sanctioned by EPA for use in predicting future air quality in the state-prepared implementation plans.

The dispersion model employed in AQDM is a simplified variant of the plume model described earlier.

AQDM also needs values for mixing depth; but these are not entered directly. Rather, they are estimated from stability information. High stability yields low mixing depth. Stability estimates are in turn derived from windspeed-cloud-cover relationships as described earlier.

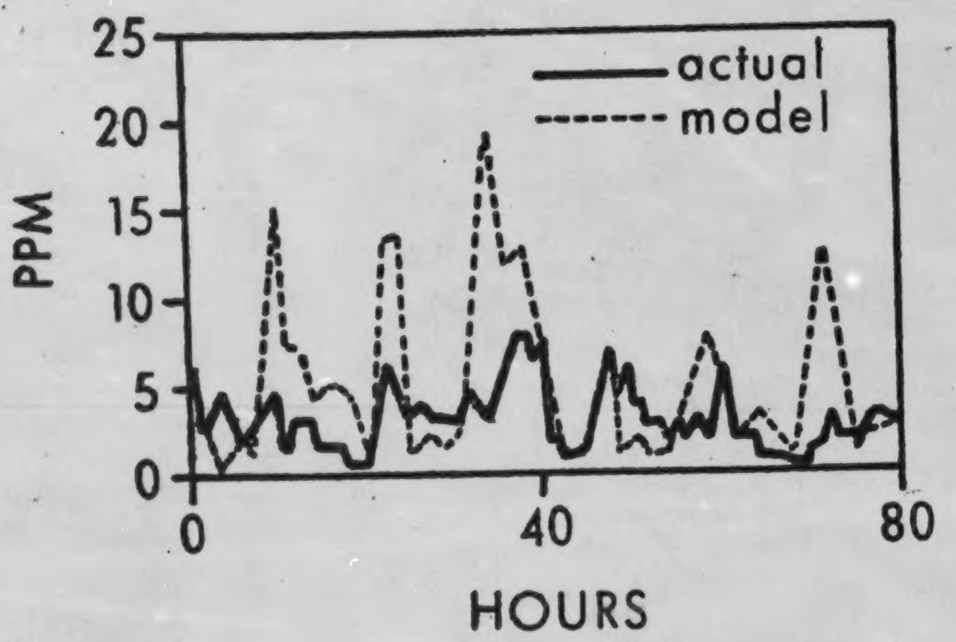


Figure 19: Comparison of measured and calculated concentration for CO with plume model.

AQDM then combines groups of short-term meteorological measurements into weighted annual averages. In all, AQDM computes concentrations for each of 480 combinations of weather conditions; comprised of 16 wind directions, six wind speed classes, and five stability classes. The frequency of occurrence of each combination is then determined. Finally, the mean annual concentration is determined as the frequency-weighted sum of concentration for each set of conditions.

Now that you have some idea of what goes on inside AQDM, let's get back to our comparison of computed versus measured results (6). Figure 20 is a plot of sulfur dioxide concentrations in Chicago. Results calculated via AQDM on the horizontal axis are compared with monitored observations on the vertical axis. Calculated, horizontal; observed on the vertical. If AQDM correctly predicted the observations, the results would lie on a 45 degree line as shown in Figure 20. As you can see, very few of the points do. In the extreme, at point A, the model calculated a concentration of 500 for an observation of less than 100. We noted this same-sized error on occasion when the standard plume model was used for a short-term comparison.

AQDM doesn't give-up here. The next step is calibration. Ideally, a straight line or simple curve could be fitted which passed through or close to each of the data points. The linear calibration line for the Chicago data is also shown in Figure 20. In mathematical terms, the equation for the calibration line is:

$$\begin{pmatrix}\text{Observed}\\ \text{concentration}\end{pmatrix} = 32 + 0.26 \begin{pmatrix}\text{Calculated}\\ \text{concentration}\end{pmatrix}$$

Note that 32 is the intersection with the vertical axis in Figure 20. This point represents the concentration in the air when emissions are zero, in other words, the background concentration. So far, so good; one would expect to have to add in a background or incoming concentration to the plume-calculation results.

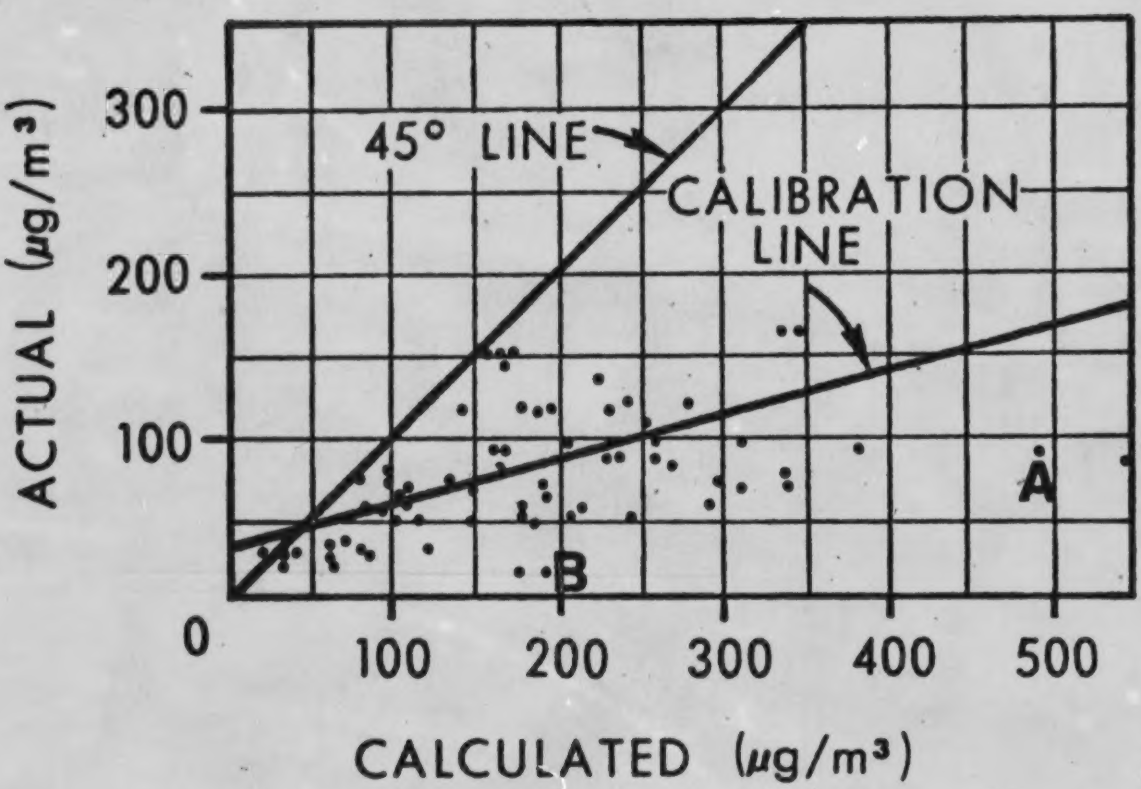


**Figure 20:** Comparison of actual versus calculated $SO_2$ concentration for Chicago.

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Unfortunately, this regression-derived calibration line, with its correction for background concentration and whatever else, still only explains 27% of the variation between the observed and calculated concentrations. In fact, some data points lie closer to the rigorous 45 degree line than to the calibration line.

Yes, overall agreement is increased a little by the calibration correction. For example, at point B a calculation of 200 is revised to 80, but in this vicinity, observed concentrations of from 20-150 exist. No single calibration line can correct for this kind of variation. The accuracy of AQDM even after calibration is about the same as that for the calibrated box model or for the short-term plume model.

AQDM does have at least one real plus. It automatically translates annual-average-concentrations into higher maxima expected for shorter time periods. You can get an automatic indication of how your plan relates to once-a-year maxima for one hour or for other short-term periods.

So much for plume models. The puff model represents a still-higher level of sophistication. Is it worth the extra time-consuming complexity (9)? You probably can guess the answer. The puff model was the one which emitted pollutants in discrete bundles which could be made to respond to conditions downwind from the stacks. Clearly, this approach is the more rigorous. For the sake of brevity, let's compare both the plume and the puff results with actual observations. Figure 21 provides a comparison using sulfur dioxide concentrations obtained from a Chicago monitoring station (9). The puff model seems to incorporate the same flaws as the plume model. The two models agree with each other better than with the observations.

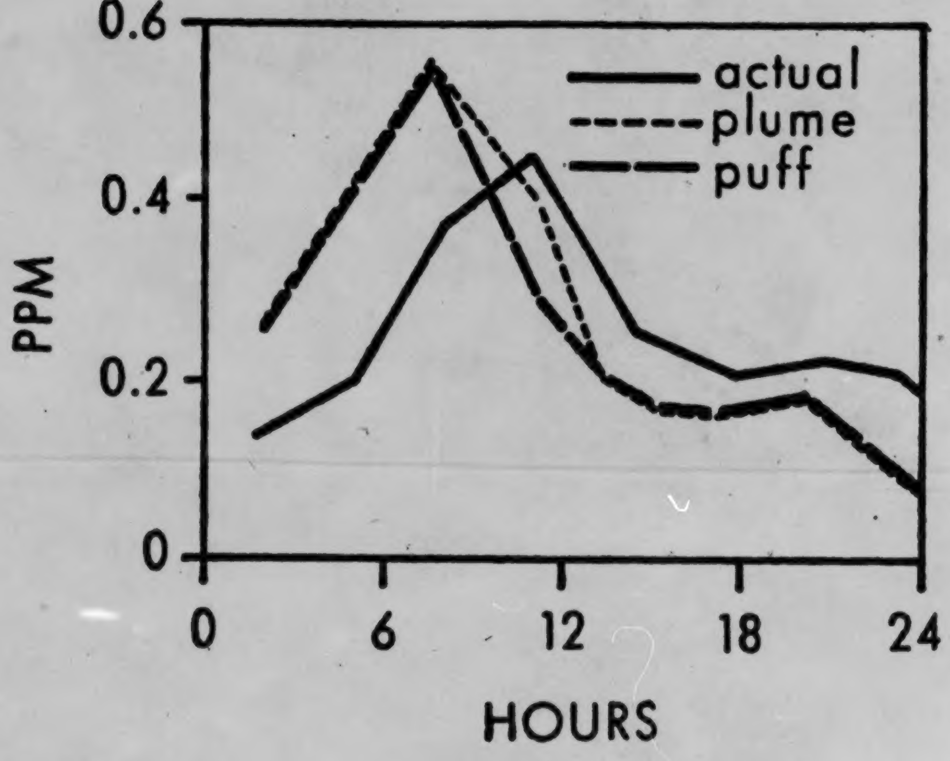

Figure 21: Comparison of plume and puff models with actual concentrations of $SO_2$ (Monitoring Station A).

Let's try it again for another monitoring station (Fig. 22). The puff model came closest to the daily maximum, but it overestimated just previous to the maximum. Overall, it's difficult to say which came closest to the observed results, but based upon this extremely limited evidence, there presently seems little real advantage of the puff model over the less complex plume model. Perhaps the situation will change when we can obtain better time-dependent emission data and urban meteorology data at more locations; but for now there seems little justification to warrant use of the puff model. To prove the point, Argonne National Laboratory, which developed the puff model, presently relies almost exclusively on the AQDM alternative.

Large errors remain, whichever model is used; but in fairness to both the plume and puff models, it should be noted that the time period used in the comparison is very short. The agreement with observation seems to indicate that we now know not enough, but still quite a bit, about urban dispersion.

I couldn't justify the time-consuming complexity of the puff over the plume model. So if simplicity is a virtue, why not go all the way and employ the box model exclusively? Clearly the box model breaks down when it comes to large point sources. The box model gives a constant concentration everywhere.

On the other hand the plume model would yield a distribution of concentration something like Figure 23. Note that these differences would decrease as the time period increases, because during the course of a year, in most locations, wind directions would vary

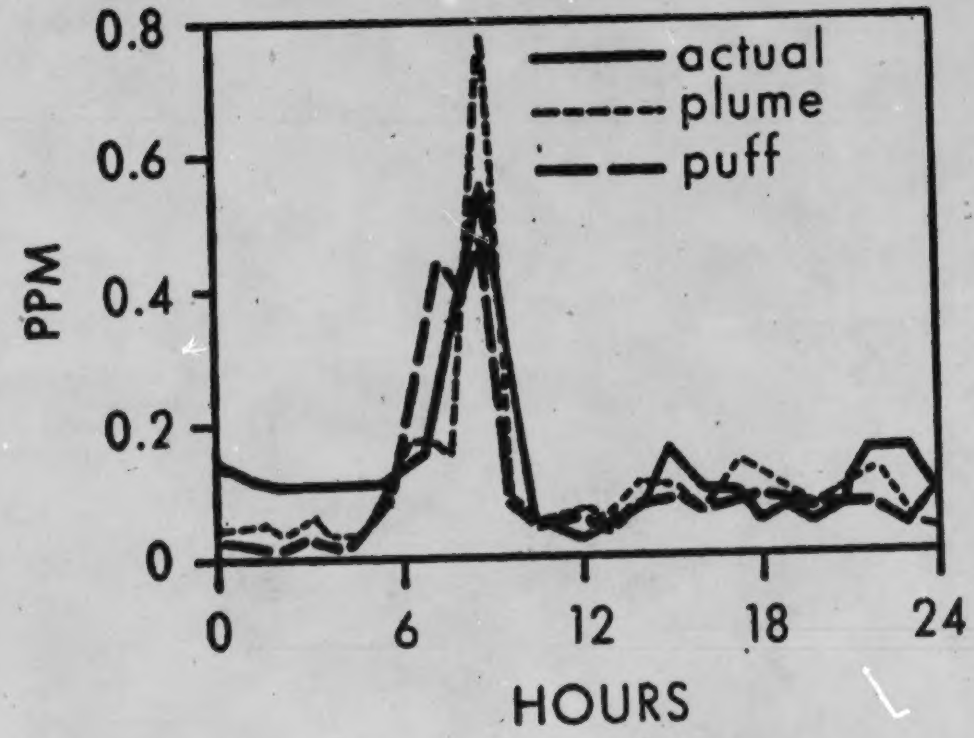

Figure 22: Comparison of plume and puff models with actual concentrations of $SO_2$ (Monitoring Station B).

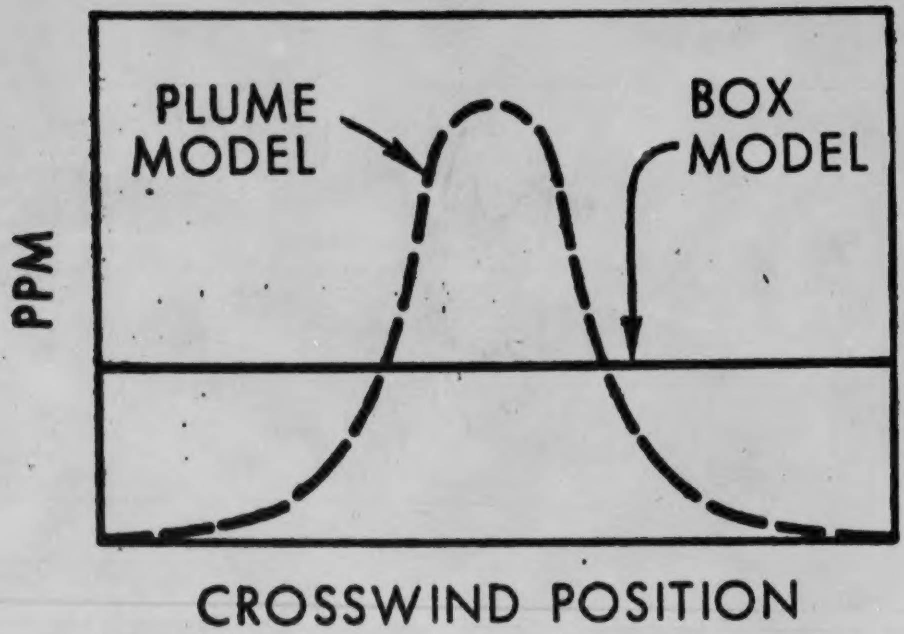

Figure 23: Comparison of box and plume model concentrations.

by 360 degrees. Likewise, windspeeds, stabilities and mixing depths would also be expected to vary markedly. When AQDM weights and sums all the 480 meteorological combinations, the result can very well resemble the crude box-model approximation.

EPA recognizes this situation. Remember, I said AQDM is one of two sanctioned methods for predicting future air quality. The other method, called "roll-back," simply assumes that any reduction in emissions immediately will be reflected as a directly proportional reduction in ambient pollutant levels, all without consideration of source-receptor geometry or the urban meteorology. This conclusion can be derived from the box model with everything held constant except C and Q. Any reduction in the emission term (Q) is directly reflected in the ambient concentration term (C).

## CONCLUSION

Our inability to identify an always accurate model leaves us facing quite a dilemma. Clearly, the best or most cost-effective way to manage air quality is to segregate sources from receptors through judicious land use control. Then I question whether we have the tools to adequately define how far from and in what direction receptors should be located from sources.

My concerns lie not so much with the models, but with the data which goes into them. Let's return to this calibration procedure. In actuality it is necessary to calibrate all the model types, whether box, plume or puff. Even if the calibration procedure worked, we would have no idea what was at fault. It could involve errors in anything I've talked about; the wind speeds or direction, mixing heights, stability, plume rise, background concentrations, recirculation, pollutant degradation, emission strength, all in addition to any shortcomings in the models themselves.

Particularly disconcerting would be a differential error involving the handling of different kinds of emissions. For example, in Chicago, particulates emanate from large point sources with high stacks and in much smaller amounts from coal-burning furnaces in apartments and commercial buildings which have low stacks. Despite the much lower emission rates, AQDM studies indicate that it is the coal-burning apartments and commercial buildings which are chiefly responsible for Chicago's high ambient particulate levels. This apparent self-pollution phenomenon has led the state and federal environmental officials to propose that residential-commercial coal burning be banned here (12). I do tend to believe the results generated by AQDM, but a lake breeze, or an urban heat island, or who knows what else, could be coloring the results, say by understating the contribution of the coal-burning power plants with their high stacks. The calibration of AQDM would simply mask such an error.

Let's get back to models. The most sensitive variable in plume and puff models is stability. Small changes in stability cause big changes in plume-dispersion behavior. Unfortunately, this is the most difficult variable to quantify, particularly in the presence of urban heat islands, lake breezes, and so on. If the situation is deemed hopless, you must revert to the rollback method and forget about optimizing source-receptor relationships.

I am not quite that pessimistic. Both box and plume models can be useful. When used appropriately for long-term assessment of air quality they can be accurate usually within a factor of two of actual air quality. When model results indicate that a given strategy will produce air quality benefits in excess of that amount, that is, if the strategy would reduce pollutant levels by more than one-half, we can proceed to a cost-benefit analysis with considerable confidence. For smaller reductions, well, we take our chances.

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# CHARACTERIZATION OF ENVIRONMENTAL HAZARDS:

# DISPERSAL OF POLLUTANTS IN WATER

G. R. Brenniman

## INTRODUCTION

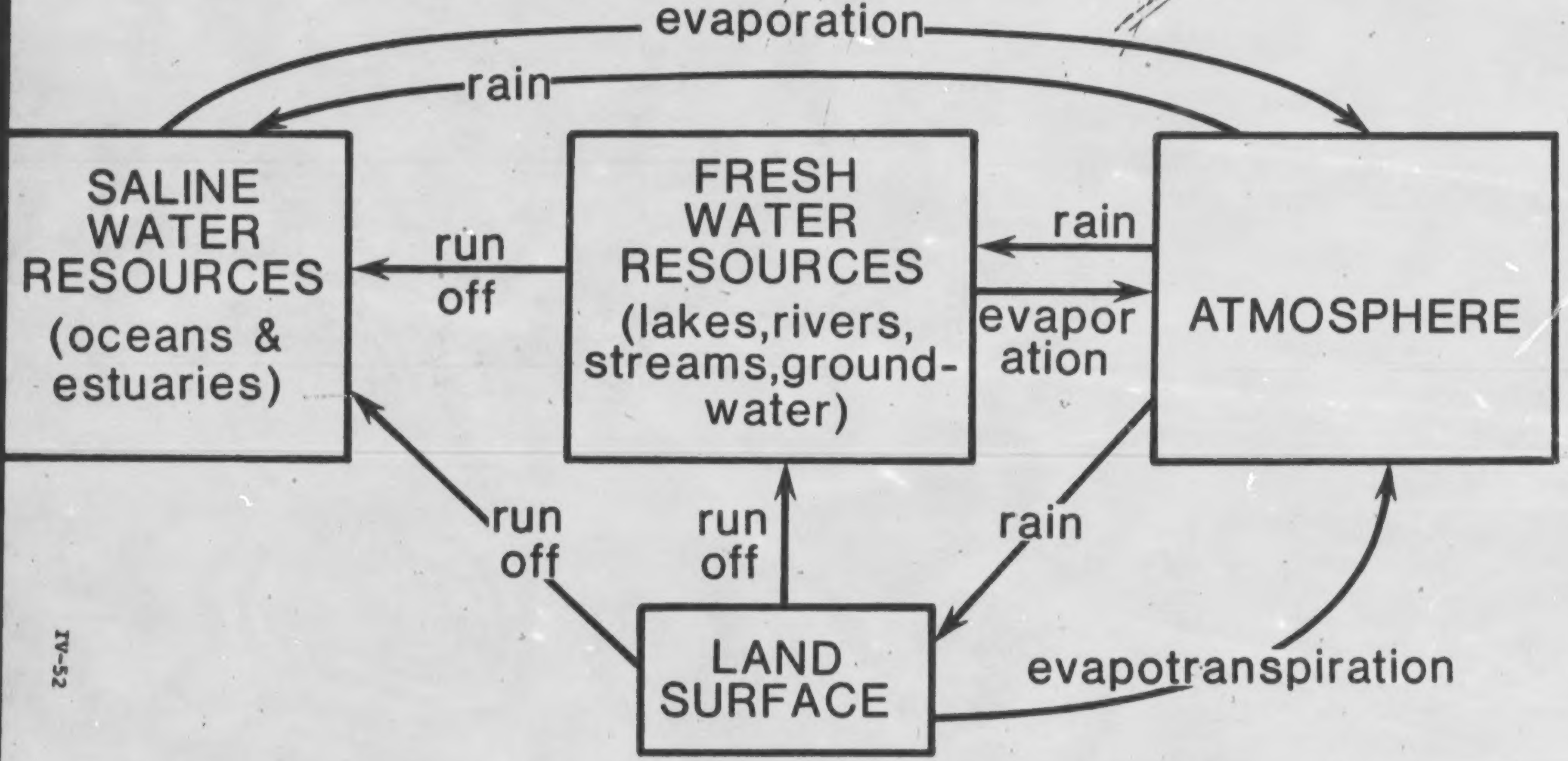
Before I discuss "Dispersal of Pollutants in Water," I would like to give a brief background of water quality management and models.

First of all, in discussing any water quality management problem we immediately realize that we have a finite amount of water that is being circulated by a water transport system--The Hydrologic Cycle (Fig. 1): While this water is circulating through the hydrologic cycle, its quality is continually changing. For instance, the quality of natural bodies of water like lakes, rivers, oceans, etc., is affected by natural phenomena associated with the geophysical, hydrological and meteorological characteristics of the drainage basin. As it rains, water picks up such natural things from the atmosphere as gases ($CO_2$, $O_2$, $N_2$), dust, microorganisms, pollen, etc. Once the water hits the ground it can pick up soil particles, vegetation and additional microorganisms. This surface runoff moves to the receiving waters, or the water can infiltrate to the groundwater supply, picking up dissolved minerals dependent primarily upon the geophysical and meteorological conditions present.

Besides the natural quality change to water as it moves in the hydrologic cycle, water also changes in quality from such man-made conditions as industrialization, urbanization and agricultural practices. Although some of these conditions consume water, most of it is returned for reuse. Receiving water systems have some natural capacity to dissipate certain pollutants. However, if this natural purification capacity is exceeded, the returned wastewaters may contain concentrations of constituents that will degrade the quality of the water to a point that may prevent its usage for public water supplies, recreation, commercial fishing, agricultural irrigation, freshwater

Figure 1

## THE HYDROLOGIC CYCLE


evaporation
rain
SALINE WATER RESOURCES (oceans & estuaries)
FRESH WATER RESOURCES (lakes,rivers, streams,groundwater)
ATMOSPHERE
run off
rain
evaporation
run off
run off
rain
LAND SURFACE
evapotranspiration

and marine aquatic life, and industrial utilization. Therefore, an example of a simple water quality management system in relation to a specific water use is shown in Figure 2.

Mathematical models can be used to predict the dispersion and concentration of pollutants in a receiving water from various inputs. As such they can tell us if the natural purification capacity has been exceeded. Models may be employed to determine a specific water quality use by analyzing past data, assessing present conditions, and projecting future needs.

Since there are differences in the geomorphological and hydrological characteristics of the many kinds of receiving waters (i.e., streams and creeks, rivers, estuaries, lakes, oceans, etc.), particular models are required for each of these systems.

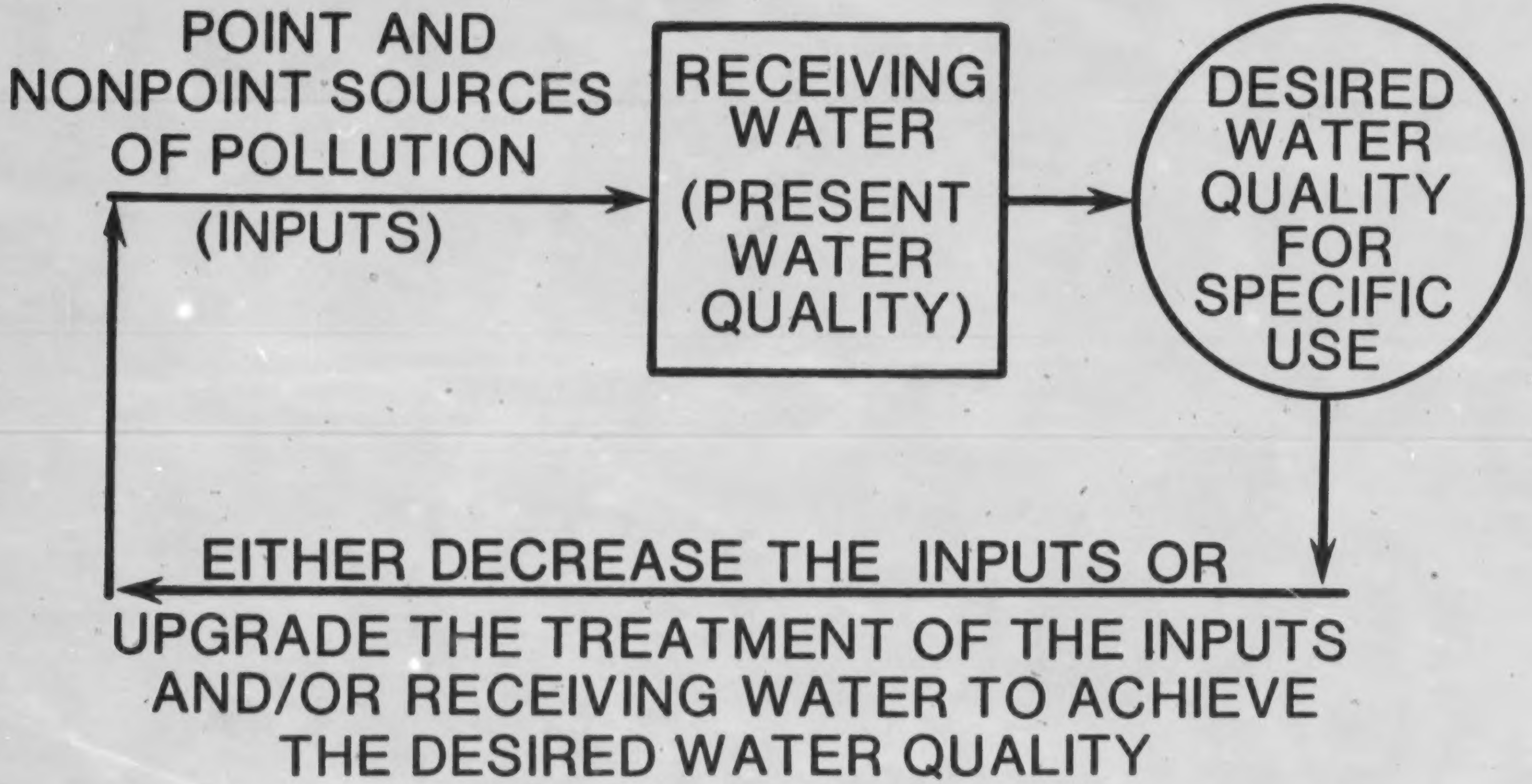
Today I am going to only address the dispersion of pollutants in rivers and small lakes and ponds. First of all, let us consider a single system water quality model for a river or stream. A single system is defined as a system where there is a direct functional relationship between a pollutant in the effluent and the concentration of that material in the stream. The pollutant in the effluent can either be termed conservative or non-conservative. A conservative pollutant has little or no decay with time, nor does it disappear from the system by settling, adsorption, or any other means. Examples of conservative pollutants would include total dissolved solids, chlorides, some pesticides, and heavy metals. In contrast, non-conservative pollutants, like biochemical oxygen demand (BOD) and coliform bacteria, do decay with time.

## PLUG FLOW

In constructing a water quality model for a single system, the concepts of plug flow and/or continuous flow stirred tank reactor

Figure 2

## SIMPLE WATER QUALITY MANAGEMENT SYSTEM


POINT AND NONPOINT SOURCES OF POLLUTION
(INPUTS)
RECEIVING WATER
(PRESENT WATER QUALITY)
DESIRED WATER QUALITY FOR SPECIFIC USE
EITHER DECREASE THE INPUTS OR
UPGRADE THE TREATMENT OF THE INPUTS AND/OR RECEIVING WATER TO ACHIEVE THE DESIRED WATER QUALITY

(CFSTR) can be employed. First of all, let us consider the application of the plug flow concept to a river or stream with the following assumptions (Fig. 3):

1. The pollutant is evenly distributed over the river cross section, A.
2. There is no mixing of the pollutant in the axial direction, X.
3. There is a single pollutant source and a single pollutant.
4. For non-conservative pollutants, first order reaction kinetics are used, so that the rate of change $\frac{\Delta C}{\Delta t}$, of any pollutant is directly proportional to the concentration of that material, C, at time, t; $\frac{\Delta C}{\Delta t} = - KC$, where K is the first order reaction rate constant.

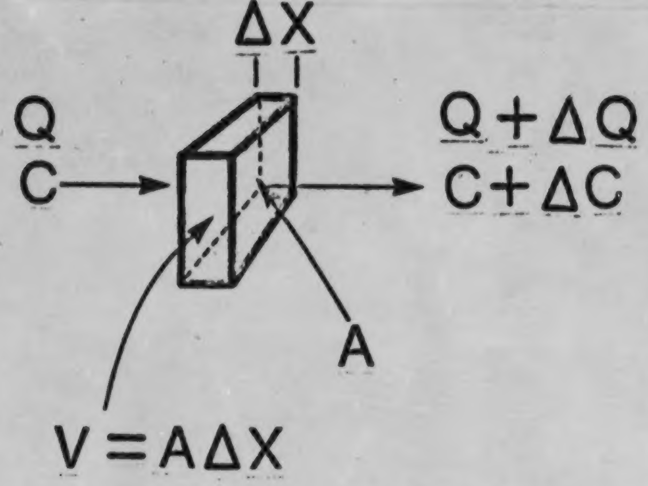
Therefore, a plug flow concept can be used to calculate the concentration of a conservative or a non-conservative pollutant at any point on a river at time t as long as the above four assumptions are taken into consideration. A mass balance equation for a particular pollutant can be written as follows to determine what this concentration will be (see Appendix for dimensions of all symbols used throughout text):

$$\text{mass}_{in} - \text{mass}_{out} - \text{reaction} + \Sigma(\text{sources}) - \Sigma(\text{sinks}) = \text{accumulation} \tag{1-1}$$

The change in mass is equal to the net difference between the inflow and the outflow taking into consideration the decay of non-conservative pollutants (reaction term) and the sources and sinks. Sources and sinks are composed of the following several components:

1. A point source (i.e., a waste discharge)--will be incorporated through the boundary condition at $X = 0$; where the concentration, $C$ = the initial concentration, $C_0$, and $Q$ = river flow. As a result, $C_0 = \frac{Q_u C_u + Q_w C_w}{Q_u + Q_w}$ where u = upstream and w = waste. If $Q_w C_w >> Q_u C_u$, and let $Q_w C_w = W$, then $C_0 = \frac{W}{Q}$.

Figure 3. Plug flow model



Q = RIVER FLOW

A = CROSS-SECTIONAL AREA

V = VOLUME [CROSS-SECTIONAL "SLICE" OF THICKNESS (ΔX) OF RIVER]

C = CONCENTRATION OF POLLUTANT ENTERING THE UPSTREAM FACE OF "V" IN A TIME INTERVAL Δt

2. A distributed source or sink that inputs material and flow along the length of the stream such as runoff and suspended solids; therefore, mass(added)/time = $C' \frac{\Delta Q}{\Delta X} \Delta X$, where $C'$, for example, is the concentration of the runoff, and $\frac{\Delta Q}{\Delta X}$ represents the change in river volume due to runoff for a particular stretch of river, $\Delta X$.

3. A distributed source or sink along the bottom or sides of a river from a previous input as from benthal deposits; therefore, $\frac{\text{mass(added or subtracted)}}{\text{time}} = S_d A \Delta X$, where $S_d$ is the amount of the pollutant that is added or subtracted from the river.

The mass balance of equation (1-1) expanded then becomes:

$$QC - (QC + \frac{\Delta QC}{\Delta X}\Delta X) - K\tilde{C}A\Delta X + C'\frac{\Delta Q}{\Delta X}\Delta X \pm \Sigma S_d A\Delta X = A\frac{\Delta C}{\Delta t}\Delta X \quad (1\text{-}2)$$

where

Q = river flow

C = concentration of pollutant from point source

A = cross-sectional area

X = "slice" of thickness of river

K = decay rate constant

$$\tilde{C} = \frac{C + (C + \frac{\Delta C}{\Delta X}\Delta X)}{2}$$

$C'$ = the concentration of the runoff

$S_d$ = the distributed source or sink from a previous input

Then, if equation (1-2) is divided by $\Delta X$, assuming that $\bar{C} \approx C$ (for a small $\Delta X$) and letting $\Delta X$ and $\Delta t \to 0$, the general plug flow equation (1-3) for a single pollutant is:

$$\frac{\partial C}{\partial t} = \left(-\frac{C}{A} + \frac{C'}{A}\right)\frac{\partial Q}{\partial X} - \frac{Q}{A}\frac{\partial C}{\partial X} - KC \pm \Sigma S_d \quad (1\text{-}3)$$

Under steady state conditions, $\frac{\partial C}{\partial t} = 0$; equation (1-3) becomes:

$$\left(-\frac{C}{A}+\frac{C'}{A}\right)\frac{dQ}{dX}-\frac{Q}{A}\frac{dC}{dX}-KC \pm \Sigma S_d = 0 \qquad (1\text{-}4)$$

Equation (1-4) can be used for both conservative and non-conservative pollutants under steady state conditions, remembering that $K = 0$ for the conservative pollutant. Equation (1-4) may be difficult to solve analytically over extended river stretches because mathematical equations are not available for term $\frac{dQ}{dX}$. Therefore, a more practical way to address the problem would be to break the river up into reaches. For example, equation (1-4) can be easily changed to fit a step-wise solution if the following assumptions are made: assume that Q is not a function of X and that there are no distributed sources or sinks, then equation (1-4) becomes:

$$-\frac{Q}{A}\frac{dC}{dX}-KC = 0 \qquad (1\text{-}5)$$

Then, if $\frac{Q}{A} = \nu$

$$\frac{dC}{dX} = -\frac{KC}{\nu} \qquad (1\text{-}6)$$

Integration of equation (1-6) results in equation (1-7):

$$C = C_0\, e^{-\frac{K}{\nu}X'} \qquad (1\text{-}7)$$

where

$C$ = downstream concentration

$C_0$ = initial concentration at time = 0

$X'$ = downstream distance

The biggest danger to this approach is that it cannot be applied over too long a distance in the river because K may change and the velocity really is not a constant.

If the pollutant is conservative, then equation (1-7) would be simplified to:

$$C = C_0 \text{ (because } K = 0) \qquad (1\text{-}8)$$

Figure 4 pictorially illustrates what happens to a conservative and non-conservative pollutant entering a river from three outfalls (segment a) and dividing the river into reaches. Segment b shows the increase in the rate of flow from the input at outfalls $X_1$, $X_2$, and $X_3$. Segments c-e also show a decrease in non-conservative pollutant due to dilution and decay. Segment f shows a summation on the conservative pollutant from the individual inputs at outfalls $X_1$, $X_2$, and $X_3$. The concentration would not fall until the flow was increased downstream from an input like a tributary that did not contain any amount of this pollutant (this situation is represented by a question mark). The non-conservative pollutant in segment f is also summed showing first an increase in concentration and then a decrease due to decay.

An example of a steady state non-conservative pollutant is the input of fecal coliforms into the river (as shown in Fig. 4) at outfalls $X_1$, $X_2$, and $X_3$. Outfall $X_1$ has zero travel time because this is the first point of pollution; $X_2$ has .273 days travel time from $X_1$; and $X_3$ has .213 days travel time from $X_2$. Travel time is the amount of time it takes a pollutant to travel down the river from the outfall ($t = \frac{X}{v}$). The flow rates at $X_1$, $X_2$, and $X_3$ are 304, 324, and 327 cfs, respectively.

In addition, assume that K = 1.023 and that C, the concentration of fecal coliforms can be calculated from the following relationship (steady state non conservative pollutant);

$$C = C_0 e^{-\frac{K}{v}X'} = \frac{(81.7(P.E.)\, e^{-\frac{K}{v}X'}}{Q} \qquad (1\text{-}7)$$

where P.E. = Population Equivalent (a term used to represent large numbers of bacteria), Q is in cfs and 81.7 is a conversion factor such that C is in coliforms per ml. Compute the fecal coliform concentrations per ml at outfalls $X_1$, $X_2$, and $X_3$, if the P.E. discharge at $X_1$ is 144.3, at $X_2$ is 432.8, and at $X_3$ is 11.8.

Solution:

The concentration of fecal coliforms at $X_1$ is:

$$C = \frac{(81.7)(144.3)}{(304)} e^{-(1.023)(0)}$$

$$C = \underline{38.9} \text{ fecal coliforms per ml at } X_1$$

at $X_2$:

$$C = \frac{81.7}{324} \left( \begin{array}{l} \text{Coliforms remaining from} \\ \text{discharge at } X_1 \end{array} + \begin{array}{l} \text{Coliforms discharged} \\ \text{at } X_2 \end{array} \right)$$

$$C = \frac{81.7}{324} \left( 144.3 \, e^{-(1.023)(0.273)} + 432.8 \right)$$

$$C = \underline{136.7} \text{ fecal coliforms per ml at } X_2$$

at $X_3$:

$$C = \frac{81.7}{327} \left( \begin{array}{l} \text{Coliforms remaining from} \\ \text{discharge at } X_1 \end{array} + \begin{array}{l} \text{Coliforms remaining} \\ \text{from discharge at } X_2 \end{array} + \begin{array}{l} \text{Coliforms discharged} \\ \text{at } X_3 \end{array} \right)$$

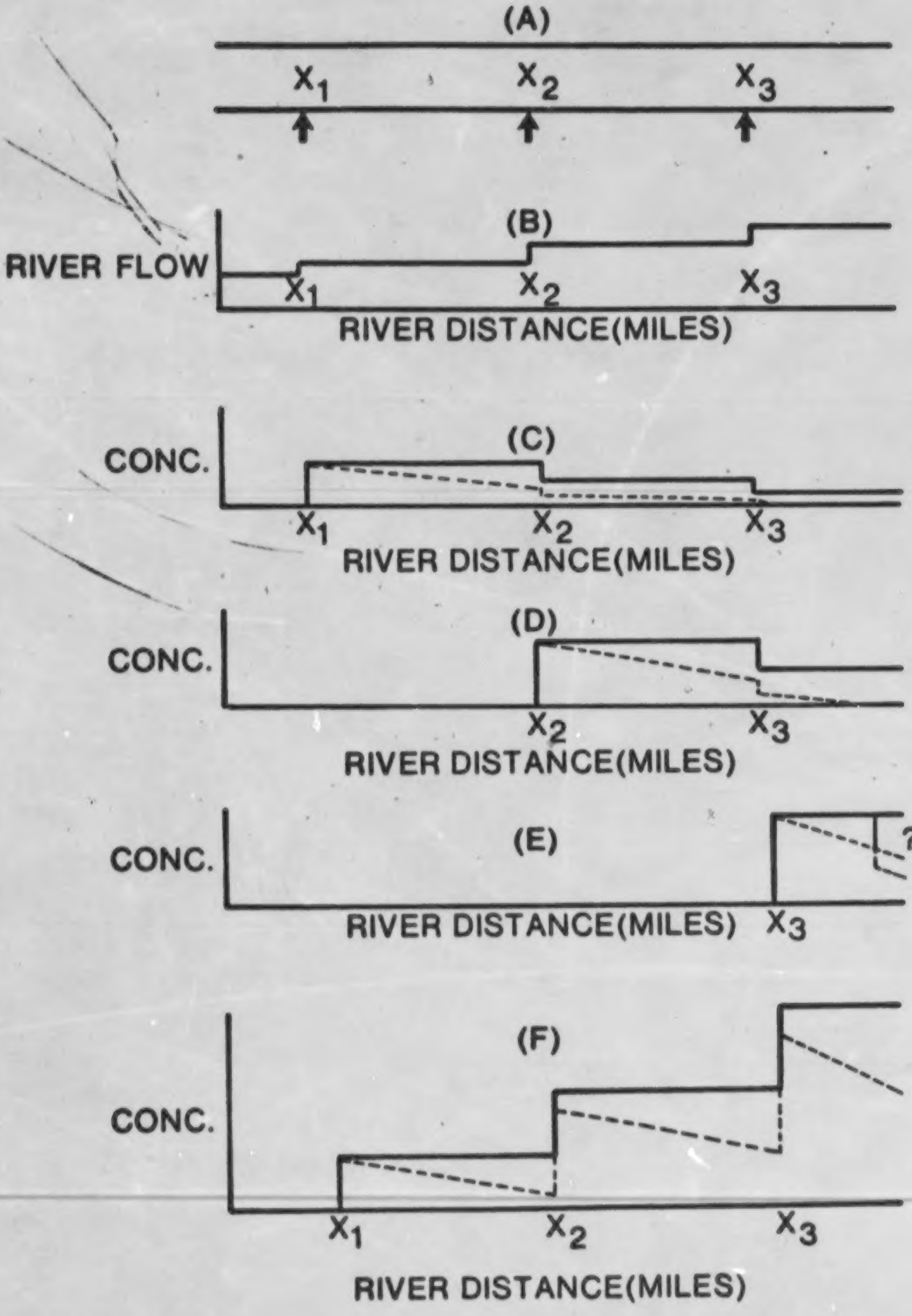
$$C = \frac{81.7}{327} \left( 144.3 \, e^{-(1.023)(0.486)} + 432.8 \, e^{-(1.023)(0.213)} + 11.8 \right)$$

$$C = \underline{111.8} \text{ fecal coliform per ml at } X_3$$

Note that the decay of number of fecal coliform is a function only of travel time in the river, and not of river volume. This example illustrates the stepwise application of the relationship for a non-conservative pollutant discharged from a point source at steady state flow for a receiving water for which the flow rate is not constant.

Figure 4

## FATE OF CONCENTRATION OF CONSERVATIVE (SOLID LINE) AND NON-CONSERVATIVE (BROKEN LINE) POLLUTANTS ENTERING DIFFERENT REACHES OF A RIVER.



An equation can now be developed which includes the contribution to the concentration C from the distributed source, $S_d$, as well as the point discharge, $C_0$. Therefore, if $S_d$ is included as a distributed source and Q and A are constant, the equation becomes:

$$\nu \frac{dC}{dX} + KC = S_d \tag{1-9}$$

The solution to equation (1-9) under the condition that C (at X = 0) = $C_0$ is:

$$C = \frac{S_d}{K}\left(1 - e^{-\frac{K}{\nu}X}\right) + C_0\, e^{-\frac{K}{\nu}X} \tag{1-10}$$

The distributed source effect is zero at the outfall and contributes to the waste concentration as the point source input moves downstream.

The general solution of equation (1-4) can be represented as follows (5):

Let $\xi = \frac{1}{A}\ \frac{dQ}{dX}$ and $\frac{Q}{A} = \nu$

Then equation (1-4) becomes:

$$(-C+C')\,\xi - \nu \frac{dC}{dX} - KC \pm \Sigma S_d = 0 \tag{1-11}$$

Let $$I(X) = \int_0^X \left(\frac{C'\xi + \Sigma\, S_d}{\nu}\right) e^{\Phi(X)}\,dX$$

$$\Phi(X) = \int_0^X \left(\frac{K + \xi}{\nu}\right) dX$$

And the solution of equation (1-11) is:

$$C = \left(C_0 + I(X)\right) e^{-\Phi(X)} \tag{1-12}$$

The major problem with this general solution (equation 1-12) is that it is hard to come up with analytical relationships for $\xi$ and $\Phi(X)$ which can be integrated. As a consequence, the step-wise solution may have to be used. The use of computers in the step-wise solution will reduce the laboriousness of the multiple calculations.

Besides dealing with a single system river water quality model, conditions arise where a coupled system water quality model has to be employed. A coupled system refers to a stream quality parameter being forced by the output of a preceding biochemical or physical reaction. A classic example of a coupled system is the effect of biochemical oxygen demand (BOD) on dissolved oxygen (DO) concentration. A second example of a coupled system is the discharge and dispersal of polyphosphate in a river which forces the production of orthophosphates by the chemical process of hydrolysis.

The modeling procedure for steady state coupled systems is identical to that of steady state single systems. A mass balance equation is written around a "slice" of river and all sources and sinks of the coupled system are considered. The plug flow concept is utilized again, as well as first order reaction kinetics. First of all, an equation is developed for a coupled system assuming a steady state, constant system parameters and no distributed sources or sinks.

The following pair of equations represent a coupled system of BOD and DO, respectively:

$$\nu \frac{dC_{BOD}}{dX} + K_a C_{BOD} = 0 \qquad (1\text{-}13)$$

$$\nu \frac{dC_{DO}}{dX} + K_d C_{DO} - K_a C_{BOD} = 0 \qquad (1\text{-}14)$$

where:

$C_{BOD}$ = the BOD concentration

$C_{DO}$ = the dissolved oxygen concentration

$K_d$ = the reaeration coefficient

$K_a$ = the deoxygenation coefficient

The $K_a$'s in equations (1-13) and (1-14) don't necessarily have to be the same, but in most cases they are because the data isn't available to assume otherwise. For the boundary condition:

$$C_{BOD} = C_{BOD_o} \text{ at } X = 0, \; C_{BOD} = C_{BOD_o} e^{-\frac{K_a}{\nu} X}$$

which substituted into equation (1-14) becomes:

$$\nu \frac{dC_{DO}}{dX} + K_d C_{DO} = K_a C_{BOD_o} e^{-\frac{K_a}{\nu} X}$$

Doing likewise for the boundary condition

$$C_{DO} = C_{DO_o} \text{ at } X = 0$$

the solution for the coupled system is

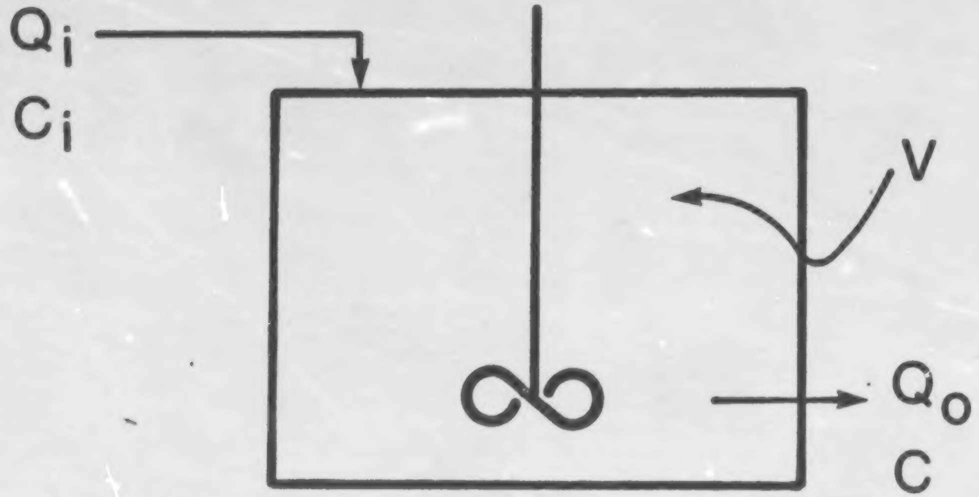
$$C_{DO_o} = \frac{K_a C_{BOD_o}}{K_d - K_a} \left[ e^{-\frac{K_a}{\nu} X} - e^{-\frac{K_d}{\nu} X} \right] + C_{DO_o} e^{-\frac{K_d}{\nu} X} \qquad (1\text{-}15)$$

## CONTINUOUS FLOW STIRRED TANK REACTOR (CFSTR)

Up to now, a plug flow model has been utilized to predict the concentration of pollutants in a receiving water. A second type of model that can be employed is the CFSTR (Fig. 5). This type of model can be applicable to small lakes, ponds, shallow reservoirs and sedimentation basins where the system is completely mixed. Therefore, this model would not work well for large lakes, oceans or estuaries.

Figure 5

# CONTINUOUS FLOW STIRRED TANK REACTOR
## (CFSTR)



WHERE: $Q_i$ = FLOW IN

$Q_o$ = FLOW OUT

$C_i$ = CONCENTRATION IN

C = CONCENTRATION OUT (SAME AS CONCENTRATION WITHIN THE REACTOR)

V = VOLUME OF THE REACTOR

The mass balance for a particular pollutant entering the CFSTR can be described as follows:

$$\text{mass}_{in} - \text{mass}_{out} - \text{reaction} + \Sigma\ (\text{sources}) - \Sigma\ (\text{sinks}) = \text{accumulation}$$

This description can be mathematically represented as in equation (1-16):

$$Q_iC_i - Q_oC - KCV + V\,\Sigma S_k = V\frac{dC}{dt} \qquad (1\text{-}16)$$

where:

$Q_i$ (Inlet flow rate) = $Q_o$ (Outlet flow rate) = $Q$

V = Volume of receiving water (constant)

$S_k$ = Distributed source or sink

$C_i$ = Inlet concentration

Under steady state conditions, there is no accumulation of pollutant, i.e., $V\frac{dC}{dt} = 0$. Therefore, equation (1-16) can be rewritten for steady state (equation 1-17):

$$C = \frac{QC_i}{Q+KV} + \frac{V\Sigma S_k}{Q+KV} \qquad (1\text{-}17)$$

If $\Sigma S_k = 0$, then equation (1-17) can be rewritten as:

$$C = \frac{QC_i}{Q+KV} \qquad (1\text{-}18)$$

where V is the volume in and out of the reactor (remember that for a conservative pollutant K = 0).

Finally, equation (1-16) for a non-steady state, non-coupled system, constant flow rate with boundary condition $C = C_0$ at $t = 0$ (initial concentration), and $C_i$ given, can be integrated to equation (1-19).

$$C = C_0\, e^{-\left(\frac{Q+KV}{V}t\right)} + \frac{QC_i + V\Sigma S_k}{Q + KV}\left[1 - e^{-\left(\frac{Q+KV}{V}t\right)}\right] \qquad (1\text{-}19)$$

An example of a problem involving a CFSTR is as follows:

A chemical plant is discharging 5000 lb/day methoxychlor into a river just before it flows into a small lake (V = 7000 acre-feet) at a flow rate of 10 cfs. (A) At steady state, what will be the methoxychlor concentration in mg/l in the lake, if it is being removed at a rate of $K = 0.1 \text{ day}^{-1}$? (B) How long will it take for the drinking water standard of 0.1 mg/l to be exceeded? (Methoxychlor is a chlorinated pesticide that attacks the central nervous system in humans.) (C) How long will it take for the lake to return to the drinking water standard of 0.1 mg/l methoxychlor, if the discharge from the plant is completely stopped?

(A) The steady state equation $C = \frac{Q\,C_i}{Q+KV}$ (1-18) for a CFSTR will be used to solve part (A) of this problem, with the following assumptions:

$$Q_i = Q_o = Q; \quad Q_{p(\text{plant})} << Q_i; \quad \Sigma S_K = 0$$

and

$$C_i \simeq \frac{Q_p C_p}{Q_i} = \frac{M_p}{Q_i}$$

where $M_p$ is the mass of pollutant from the plant.

Therefore;

$$C = \frac{QC_i}{Q+KV} = \frac{M_p}{Q+KV}$$

$$C = \frac{(5000 \text{ lb/day})\ (454 \text{ g/lb})\ (10^3 \text{ mg/g})}{\left[(10 \text{ ft}^3/\text{sec})\ (28.3 \text{ l/ft}^3)\ (86{,}400 \text{ sec/day}) + (.1/\text{day})\ (7000 \text{ acre-feet})\ (43{,}560 \text{ ft}^2/\text{acre}\ (28.3 \text{ l/ft}^3)\right]}$$

$C = \underline{2.56 \text{ mg/l}}$ methoxychlor in the lake under steady state conditions

(B) Equation (1-19),

$$C = C_0 e^{-\left(\frac{Q+KV}{V}\right)t} + \frac{QC_i + V\Sigma S_k}{Q + KV}\left[1 - e^{-\left(\frac{Q+KV}{V}\right)t}\right]$$

for CFSTR will be used to solve part (B) of this problem with the following assumptions and conditions:

$C = 0.1$ mg/l methoxychlor

$C_0 = 0$

$\Sigma S_K = 0$

$QC_i = M_p$

Therefore:

$$C = \frac{M_p}{Q+KV}\left[1 - e^{-\left(\frac{Q+KV}{V}\right)t}\right]$$

From part (A), $\frac{M_p}{Q+KV} = 2.56$ mg/l methoxychlor

Then, substitute into the above equation and solve for t:

$$0.1 = (2.56)\left[1 - e^{-\frac{(10)(86400) + (0.1)(7000)(43{,}560)}{(7000)(43{,}560)}t}\right]$$

t = 0.39 days for the drinking water standard of 0.1 mg/l methoxyclor to be exceeded.

(C) As in part (B), equation (1-19) will be used to solve part (C) of the problem with the following assumptions and conditions:

$C = 0.1$ mg/l methoxychlor

$C_0 = 2.56$ mg/l methoxychlor

$\Sigma S_K = 0$

$C_i = 0$

Therefore, equation (1-19) is reduced to:

$$C = C_0 e^{-\left(\frac{Q+KV}{V}\right)t}$$

Then substitute into the above equation and solve for t:

$$0.1 = (2.56)\left[e^{\frac{-(10)(86400)\ +\ (0.1)(7000)(43,560)}{(7000)(43,560)}t}\right]$$

t = 31.56 days for the lake to return to the drinking water standard of 0.1 mg/l methoxychlor.

Models dealing with the dispersion of pollutants into ocean and lake currents and into tidal estuaries utilizing the mass balance concept are also available. These models will give concentrations of pollutants that will fall somewhere in between the limits of a plug flow model and a CFSTR model. Information dealing with ocean and lake dispersal of pollutants can be obtained respectively from Brooks (1) and Csanady (2), while estuarian dispersal can be found in O'Connor (3, 4) and Thomann (5).

## CONCLUSIONS

1. Many times it is beneficial to apply both plug flow and CFSTR models to the same problem situation to see what concentration of pollutant is obtained from each model. Generally this will give the upper and lower limits of the pollutant.
2. Models dealing with dispersal of pollutants in water can be derived using a mass balance concept where $\text{mass}_{\text{in}} - \text{mass}_{\text{out}} - \text{reaction} \pm \Sigma$ (sources and sinks) = accumulation.
3. Mass balance equations that give a "general" solution many times have limited use because some of the relationships needed to solve the equations analytically are not available. Therefore, a step-wise solution over several reaches of the receiving water may have to be employed.
4. All models need to be field tested. They are only as good as the data that are being entered into them.

# APPENDIX

## DIMENSIONS OF ALL SYMBOLS USED IN TEXT

| | Symbol | Title | Dimensions |
|---|---|---|---|
| 1. | $A$ | Cross-sectional area | $L^2$ |
| 2. | $C_{BOD}$ | Biochemical oxygen demand | $M/L^3$ |
| 3. | $C$ | Concentration of material | $M/L^3$ |
| 4. | $C_{DO}$ | Dissolved oxygen concentration | $M/L^3$ |
| 5. | $K$ | First order decay coefficient | $1/T$ |
| 6. | $K_d$ | Reaeration coefficient | $1/T$ |
| 7. | $K_a$ | Deoxygenation coefficient | $1/T$ |
| 8. | $Q$ | River flow | $L^3/T$ |
| 9. | $S_d$ | Distributed source | $M/L^3T$ |
| 10. | $t$ | Time | $T$ |
| 11. | $V$ | Volume of water body or "slice" of it | $L^3$ |
| 12. | $X$ | Distance in direction of flow | $L$ |
| 13. | $\nu$ | River velocity | $L/T$ |
| 14. | $S_k$ | Source or sink for CFSTR | $M/L^3T$ |
| 15. | $C_i$ | Inlet concentration for CFSTR | $M/L^3$ |
| 16. | $C_0$ | Initial concentration in CFSTR (at $t = 0$) | $M/L^3$ |

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# EVALUATION OF PLANNING ALTERNATIVES: POLLUTANTS WITH AND WITHOUT STANDARDS

A. H. Wolff and R. A. Wadden

In recent years there has been increasing demand from scientific and public sectors to evaluate the environmental consequences of certain human directed activities. Certainly many if not most new projects for the production, distribution and the use of energy will significantly impact the environment and, therefore, would warrant such evaluative procedures. As most of you know the term commonly used to describe this type of evaluative procedure is the "environmental impact statement."

The demand for "impact statements" was an outgrowth of the "ecology" awareness of the 1960's and, in my view, has been inordinately influenced by ecologically concerned and conservation-oriented citizens and groups. As a result, impact statements were often designed principally to protect natural resources and, as suggested in a recent Science editorial*, have increasingly been used to satisfy "ecofreaks." It would seem that the definition of "natural resources" often excludes humans because environmental impact statements generally have ignored or given inadequate attention to the effects of environmental pollutants on human health. It is this deficiency -- that is, the failure to adequately evaluate the health consequences of environmental pollutants -- that is the raison d'etre of this workshop.

Factors to consider and methodologies that can be used for evaluating the health impact of pollutants associated with energy projects will be covered in suceeding discussions.

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* Science Vol. 192, No. 4239, 7 May 1976.

Although the theme of this workshop specifically relates to environmental health impacts we cannot lose sight of the real world where selection of the "best" plans or alternatives will be based not solely on health considerations but rather, at least conceptually, on various cost-benefit considerations. The word "conceptually" is used advisedly because in many instances the public health risks are ill-defined, or unknown and controversial. Consequently we can generally quantify or monetize the strictly economic aspects of a project with much greater precision and accuracy than can be done for any health risks that may be attributed to the project. Indeed in most instances it will be possible to make only very crude estimates of the types and magnitude of health effects that may be associated with environmental pollutants. But, as has been discussed, many suspect environmental pollutants cannot be assumed to be safe just because definitive evidence or precise data regarding health effects is not yet available.

Let us now consider the factors and approaches that can be used in the evaluation of the health impacts from pollutants. When analyzing a particular project which may discharge environmental pollutants, a set of alternative plans should be available for evaluation and comparison. The types of alternative plans that may be developed will depend on the nature of the particular project under consideration. For example, with a fossil-fuel burning power plant, the alternatives may include a number of geographical sites and different types of control equipment. Or in the case of a highway network, several alternate highway routes and vehicle loadings can be compared. As previously noted the selection of the "best" alternative should be based on a variety of cost-benefit factors but our discussions today will focus only on the public health criteria that should be considered in the evaluation and decision process.

In considering public health criteria it is convenient to classify pollutants of interest into two broad categories: 1. pollutants for which there are standards based on recognized health effects; and 2. pollutants generally considered to be toxic substances but for which no standards have yet been developed. Many projects, of course, may involve pollutants that fall into both categories.

## POLLUTANTS WITH STANDARDS

For pollutants with environmental standards (e.g., sulfur dioxide and carbon monoxide in air; and phenol and mercury in water) concentrations in air or water can be calculated from the predicted pollutant discharge rates and appropriate dispersion models. Human exposure models and estimates of resultant morbidity and mortality can be developed. If the calculated environmental concentrations from a given planning alternative exceed the standard, then the plan should be modified accordingly or rejected.

Even in instances where the environmental concentrations of a particular plan are below the applicable standards it will be useful to calculate the exposure levels. It is then possible to estimate the health impact to enable a comparison to be made of the relative risks of the various alternatives in order to minimize exposure of hypersusceptible population subgroups.

## POLLUTANTS WITHOUT STANDARDS

For pollutants with suspect toxic properties for which standards have not been developed, evaluative approaches similar to those discussed above can be employed. Estimates of environmental concentrations and human exposure models should be developed. However there are no "benchmarks" i.e, environmental standards or risk estimates, against which estimated environmental concentrations can be judged to be "safe" or "acceptable." Thus, it will be necessary to exercise value judgments based on estimates of the health effects that are presumed to be associated with the projected body burdens.

There are virtually thousands of inorganic and organic substances that occur as environmental pollutants and which are known to be toxic at relatively high levels of exposure in man or animals. However, we have little if any information on the toxicity of these substances as a result of ambient exposure levels. Nonetheless, most of these toxic agents are suspected of being causal factors for various chronic diseases including cancer.

The problem of quantitating the health risks and setting standards for such agents can be illustrated by the large group of environmental pollutants with carcinogenic properties. At the present time, there are no feasible methods to determine whether there is a threshold or safe level for most carcinogenic agents. Consequently, standards for suspect carcinogens cannot be established with confidence, according to scientific criteria. From the viewpoint of protecting the public, it appears reasonable to support the controversial concept that there is "no safe dose" of a carcinogen and that any level of exposure to a carcinogen should be considered toxicologically significant for man.

There is a need to develop models that conservatively estimate the cancer risk of environmental carcinogens in a way similar to that often used for ionizing radiation -a proven carcinogen at moderate exposure levels. In the case of radiation exposure, it is assumed that there is no threshold, and that there will be an excess cancer risk in an exposed population that is directly proportional to the magnitude of radiation dose received by that population. Estimates of risk at low doses are derived accordingly.

As safe levels of carcinogens for man cannot be established by application of present knowledge, the concept of "socially acceptable risk" is more realistic. This entails the weighing of the estimated risk of potentially hazardous substances in our environment against the societal benefits of the sources.

It is especially important that risk/benefit analyses of technologic advances and new products be carried out before they are launched for public use. Particular attention should be directed to the reduction or prohibition of hazardous substances in the environment that originate from sources having the least societal benefit.

In our view, environmental pollutants suspected of a causal association with serious chronic diseases should be judged "guilty until proven innocent." The alternative is to wait many years for the evidence on potentiation of the disease, at which time remedial actions would be obsolete and worthless.

# EVALUATION OF PLANNING ALTERNATIVES: HIGHWAY NETWORK PLANNING

Donna O. Farley

Transportation systems are the focal point of one of the more sophisticated planning techniques. Computerized mathematical models can simulate variations in an existing or proposed transport system, and those simulations are an integral part of transportation plan evaluations.

A transportation plan is evaluated by measuring several alternative plans against a set of pre-established objectives, such as increased mobility, improved service, economic viability and positive ecological effect. The plan which best meets the objectives is theoretically the one selected for development. The study described here was conducted to develop a methodology that could be used to quantify health impacts of alternative transportation plans, so that health impacts could become a part of transportation plan evaluation processes.

The study was performed under the auspices of the Northeastern Illinois Planning Commission (NIPC) and funded in part by a grant from the Illinois Department of Transportation (IDOT). The alternative highway plans evaluated in the study were portions of regional plans developed by the Chicago Area Transportation Study (CATS), and all traffic data used were obtained from CATS. The study is described in detail in other reports (2,8).

The computer model used in transportation planning involves a set of geographical zones identified by CATS. Trip generations, origins and destinations are identified for these zones. Within a highway network a series of nodes are located along roadway alignments, usually at intersections. The length of road between two nodes is called a link. Each link carries only one-way traffic, and it is paired with another parallel link carrying the traffic in the opposite lanes. Route mile length, vehicle volume, and average traffic speed are defined for each link and are assigned to adjacent zones thereby establishing the geographical traffic data base.

Because the purpose of the methodology was to include health impacts as a part of transportation plan evaluations, it was considered essential that it be designed to utilize the geographic base and the basic traffic data from which all the other plan evaluation measures were also derived. Both the geographic grid system established by CATS and the format of data available from them strongly influenced the design of this study.

## GENERAL APPROACH

The methodology compared the potential health impacts for each of the highway alternatives, expressed in terms of the amounts of pollutants emitted and the sizes of populations potentially exposed to them. Emission levels from freeways and arterial highways were calculated for a series of geographic zones within the study area, and these levels were related to the sizes of several hypersusceptible populations in the same zones. The emphasis was on the estimation of potential local impacts from main freeways, so the analysis was limited to corridors of land within which freeway alignments were centered.

The total population and seven hypersusceptible populations were used as the measures for potentially exposed populations. These groups were enumerated for each zone in the freeway corridors.

Carbon monoxide, lead and noise were used as the three measures of pollution generated by motor vehicle traffic. Emissions were calculated for each pollutant from traffic data and were tabulated for each zone in the freeway corridors. This was done for each alternative. Dispersion modeling to estimate air quality was not performed because of inadequate background air quality data and freeway alignment specificity.

Evaluation for potential health impacts was conducted on two levels. An overall evaluation compared total emission levels for the alternative highway plans and the numbers of hypersusceptible persons potentially exposed to pollutants generated. A specific zone analysis was also performed to identify high risk zones. High risk zones were defined as those zones that have both (a) a large incremental increase in emissions of a pollutant between alternatives and (b) a high density of the population(s) hypersusceptible to that pollutant. This analysis determined the frequency and locations where potential significant increases in highway pollutant emissions would coincide with large concentrations of susceptible persons.

## STUDY AREA - ORGANIZATION OF DATA BASE

The study area consisted of 645 square miles of land in northeastern Illinois. Within the study area the four plan alternatives used in the study (Fig. 1 through 4) had the following freeway miles:

| Alternative | Miles of existing roads | Miles of proposed roads | Total Miles |
|---|---|---|---|
| 1 | 103 | -- | 103 |
| 2 | 103 | 66 | 169 |
| 3 | 103 | 73 | 176 |
| 4 | 103 | 102 | 205 |

For each alternative,land corridors were delineated as the potential impact areas, using the freeway alignments as their centerlines. Corridors with a total width of three miles were established for proposed freeways, and one-mile wide corridors were used for existing roads. The wider corridors used for the proposed roads reflected the uncertainty of their precise locations. Corridor boundaries were drawn to coincide with CATS zone boundaries, such that every zone falling wholly or partially within a corridor width was fully

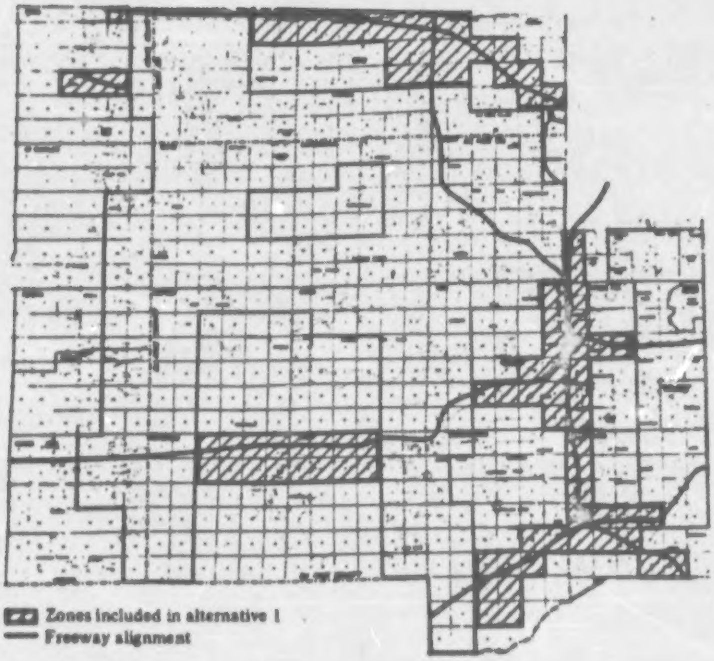

Figure 1. Alternative 1: location of existing highways.

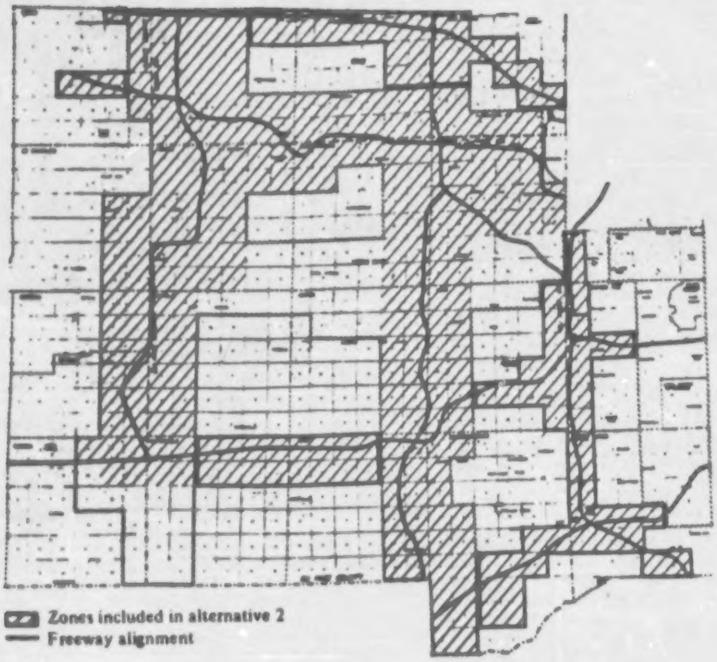

Figure 2. Alternative 2: location of existing and proposed highways.

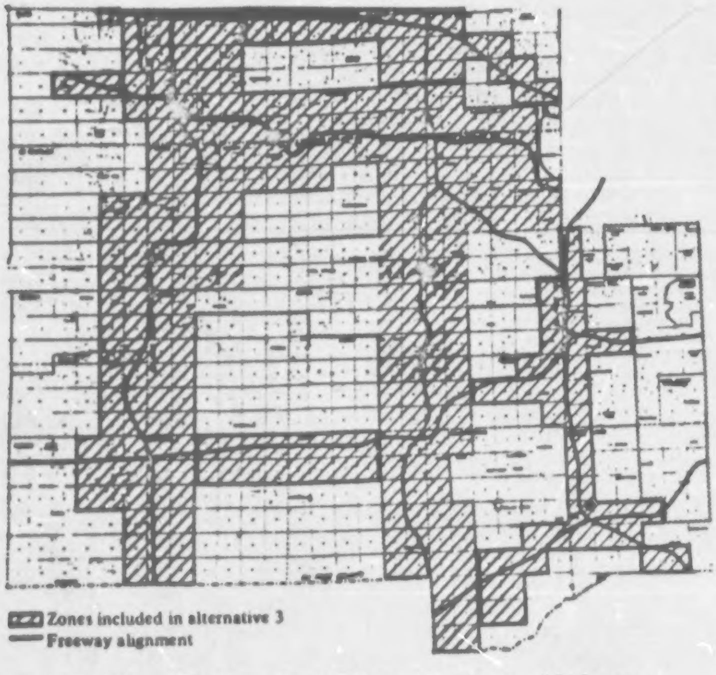

Figure 3. Alternative 3: location of existing and proposed highways.

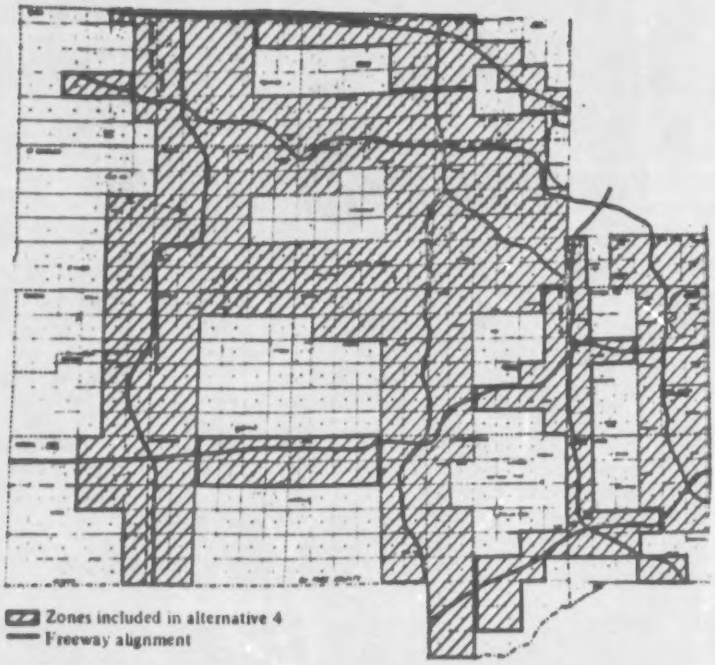

**Figure 4.** Alternative 4: location of existing and proposed highways.

included in it. Emissions and population data were developed for these zones.

After corridor zones were established, freeway links were assigned to the zones within the corridors. For the proposed corridors, links were assigned to each zone across the width of the corridor where the link was located, reasoning that its ultimate alignment might be in any one of those zones. Zones located where freeways intersected were assigned segments from both freeways, to account for the increased emissions resulting from more than one freeway. Once freeway segments were assigned to zones, their traffic data could be used to calculate freeway contributions to emission levels for the zones for pollutants being analyzed.

Transportation data were also available for arterial highways in the study area. Through a method similar to that described above, these data already had been developed by
CATS for each zone. Emissions generated by the arterial traffic were calculated, and together with the freeway emissions they provided estimates of total highway emissions for each zone in the freeway corridors.

## SELECTION OF POLLUTANTS AND EMISSION CALCULATIONS

Carbon monoxide (CO) and lead (Pb) were selected as representatives of chemical emissions from vehicular sources. They were chosen because of their obvious and documented health effects on certain populations and because vehicles contribute an important portion of total airborne quantities of both materials. Both carbon monoxide and lead can be quantified for this type of analysis because their emission factors are reasonably well defined, and they are essentially unreactive in the atmosphere. Each represents a different emission pattern, with lead being emitted in proportion to gasoline consumption, and CO emissions being dependent on the efficiency of the combustion itself.

It is interesting to note that at the time lead was selected for this study, its choice was questioned because it was not then generally considered to be an important airborne pollutant. There was substantial disagreement over the severity of health hazard posed by lead in the air when compared, for example, with hazards of lead-based paint. Added to this was a U.S. EPA regulation already establishing a timetable to reduce lead in gasoline to 0.5 gm per gallon by 1979. As of 1976, however, lead has become a criteria air pollutant because of its recognized health effects, and lead air quality standards will soon be established by the U.S. EPA. Thus, its use as an important and representative vehicular pollutant has been verified.

CO emissions were calculated for each freeway segment and for arterial traffic in each zone by the formula:

$$E_{CO} = m_v f_s \ (0.78) \tag{1}$$

where: $E_{CO}$ = CO emissions in gm, $m_v$ = vehicle miles of traffic in each segment or zone, $f_s$ = speed adjustment factor for CO emission rates, and 0.78 = CO emission factor for urban traffic in gm/vehicle mile. The emission factor was developed by CATS and was provided as part of the data obtained from them. The $f_s$ was obtained from Reference 1.

Lead emissions were calculated for each freeway segment and for arterial traffic in each zone by the formula:

$$E_{Pb} = m_v c_{Pb} \ (1/14) \tag{2}$$

where: $E_{Pb}$ = lead emissions in gm, $m_v$ = vehicle miles of traffic in each segment or zone, $c_{Pb}$ = the lead content in gasoline in gm/gal, and 1/14 = the assigned gasoline consumption in gal/mile. For this analysis a value of $c_{Pb}$ = 2.24 gm/gal was used.

Noise was selected as the third pollutant because vehicular noise is the major source of noise in most residential neighborhoods. Noise sources include tires and engine operation. Its impacts differ greatly from the chemical toxicity of the first two pollutants, varying widely and affecting a wide range of people and their activities.

Noise levels were estimated for each freeway segment using the Argonne "Hand Method for Calculation of Highway Noise Levels" (6). A $L_{50}$, which is the noise level exceeded 50% of the time, was calculated based on estimated average hourly traffic volumes from CATS data and an assumed traffic mix of 82.1% autos and 17.9% trucks.

Noise levels could not be calculated for arterial traffic because reliable average speed estimates were not available.

The freeway $L_{50}$ noise levels were converted to an index of Speech Impairment Risk (SIR) developed by K. D. Kryter and published in the Journal of the Acoustical Society of America (5). The SIR measures the percent of hearing impairment expected to occur in up to 10% of a population resulting from near daily noise doses to a given noise level over an adult lifetime (45 years) of exposure. The measure of impairment is the percentage of simple sentences heard incorrectly at 65 dBA, which is the level of everyday speech. Hearing loss is expected to occur only from exposure to noise levels above 55 dBA. Using the $L_{50}$ as the average level of noise exposure, an $SIR_{50}$ was calculated as:

$$SIR_{50} = 2(L_{50}-55). \qquad (3)$$

SELECTION AND ENUMERATION OF HYPERSUSCEPTIBLE POPULATIONS

The population groups used as high risk populations and the pollutants to which they are hypersusceptible are given in Table 1. The Pb/CO listed in the table signifies simultaneous exposure to emissions of

**Table 1.** Populations at risk.

| Susceptible population | High-risk pollutant |
|---|---|
| Total population | Pb, CO, and noise |
| Children 0–2 years old | Pb, CO, noise, and Pb and CO |
| Day-care centers | Pb, CO, noise, and Pb and CO |
| Hospital beds | CO |
| Long-term care beds | CO |
| Population over 65 years old | CO |
| Elementary-school population | Pb, CO, and noise |
| People below poverty level | Pb and CO |

both lead and CO. This indicator represents a potential synergistic effect between the two pollutants which, when both of them are present in elevated concentrations, could result in effects worse than either pollutant acting alone. Zones in which high emissions of both lead and CO occurred together were specifically identified in the analysis.

The listed populations were enumerated for each zone in all corridors in the study area. (See in this volume:"Location and Enumeration of Hypersusceptible Populations,"for a description of the demographic and high risk facilities methods used). The total population was available by mile section from NIPC data. Children 0-2 years old, the population over 65 and people below poverty level had to be estimated for each zone, using percentages obtained from census data for census tracts located in the zones (7). A listing of day care centers was obtained from the Illinois Department of Children and Family Services. Hospitals and long-term care facilities were listed in the Directory of Health Care Facilities, published by the Illinois Department of Public Health (3). Grade school listings were published in the Directory of Illinois Schools (4), and they were checked against lists obtained from individual school districts in the study area.

## EVALUATION OF THE FREEWAY PLANS

Alternative 1 consists of the existing arterial highway system and freeway network. It is used as the base case with which alternatives 2,3 and 4 are compared. In the comparison the incremental potential health impacts are evaluated for the addition of new freeway miles and alignments as defined for each alternative.

As described above, the total population and seven other hypersusceptible groups are enumerated for each zone. Their distributions within the study area are related to each highway alternative by overlaying on these population data (by zone) the emission estimates (by zone) for that alternative. The variables influencing potential increases in exposure for those populations are, therefore, the lengths, locations and intensity of traffic uses of the new freeways.

## OVERALL EVALUATION

Trends in overall emission patterns and potential exposure intensities for the whole study area are quantified in the first part of the evaluation. The analysis allows a crude comparison of the overall potential incremental impacts of alternative 2,3 and 4 as freeway plans, not yet accounting for the site-specific relationships (by zone) between emission levels and population groups.

The populations at risk for each alternative are given in Table 2. This table shows the total number of people in zones impacted by existing and new freeways for each of the alternatives. It can be seen that alternative 4, with the greatest route miles of new freeway (102 additional miles), potentially impacts almost 200,000 additional high risk persons and almost 600,000 additional total population over the base case (alternative 1). This is about twice the incremental impacts of alternatives 2 and 3.

Total study area emissions of CO and Pb for each alternative are given in Table 3. Also given are the increments in emissions of each alternative over the base case. The estimates show that an increase in total Pb emissions occurred as freeway miles increased, but did not occur for CO. Total emissions of CO stayed the same or dropped as freeway miles were added, reflecting the dependence of CO emissions on vehicle speed and combustion efficiency. Noise $SIR_{50}$ impacts could not be similarly summarized since they are measures of hearing loss risk based on local sound energy rather than mass emissions.

The initial overall study area estimates were further evaluated by describing the distribution of zones and populations within the study

**Table 2.** Enumeration of total population and hypersusceptible groups located within highway corridors.

| Population group | Alternative | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| Total population | 185358 | 449924 | 462861 | 710718 |
| *Hypersusceptibles* | | | | |
| Children 0–2 years old | 9638 | 26570 | 27271 | 39840 |
| Day-care centers | 1284 | 2898 | 2898 | 4540 |
| Hospital beds | 776 | 776 | 776 | 3129 |
| Long-term care beds | 757 | 1510 | 1655 | 2053 |
| People aged 65 years or older | 10488 | 23463 | 24494 | 45109 |
| Elementary-school enrollees | 28238 | 72919 | 74421 | 116306 |
| Persons below poverty level | 6480 | 14465 | 14890 | 26154 |

Table 3. Absolute and Incremental CO and Pb Emissions for Alternatives 1 through 4

| | CO Emissions (kg/day) | |
|---|---|---|
| Alternative | Absolute | Incremental over Alternative 1 (%) |
| 1 | 129,440 | |
| 2 | 127,060 | -2,378 (-1.8%) |
| 3 | 126,084 | -3,355 (-2.6%) |
| 4 | 126,762 | -2,677 (-2.1%) |
| | Pb Emissions (kg/day) | |
| 1 | 3,143 | |
| 2 | 3,368 | 225 (+7.2) |
| 3 | 3,390 | 247 (+7.9) |
| 4 | 3,550 | 407 (+13.0) |

area according to the emission levels estimated for them. This was done for all three pollutants, and the distributions of populations for alternatives 1 and 4 (minimum and maximum) are shown in the histograms in Figures 5 through 10. The mean CO and Pb potential emission exposures, which are population-weighted averages for all the zones included in the analysis, are also shown on the histograms.

Different patterns of potential exposure can be seen for each pollutant. Consistent with the total CO emissions for the overall study area, which do not increase with added freeway miles, the pattern of potential population exposure to CO emission levels also appears to be steady. In contrast, increased emissions of Pb as freeway miles increase result in a larger population experiencing higher Pb emissions as well as a reduction in population exposed to low emission levels. This pattern is reflected in the higher mean Pb exposure shown for alternative 4 and in the shape of the histogram.

Noise levels were only calculated for freeway traffic. Therefore, the zones and population actually experiencing freeway noise impact increases with an increase in miles of freeway. The pattern of exposure, however, is found to change as freeway miles and total exposed populations increase. Comparing alternatives 1 and 4, in Figure 9 and 10, there is some increase in population at lower $SIR_{50}$ values as well as a substantial increase at higher $SIR_{50}$ levels. The increase at low $SIR_{50}$ levels can be attributed to a drop in traffic and noise levels on existing freeways. The total traffic on new freeways accounts for the greater exposure at higher $SIR_{50}$ values. The mean $SIR_{50}$ values for the two alternatives reflect this pattern by showing similar average risks of hearing loss (from $SIR_{50}$ of 13.63% for alternative 1 to 13.56% for alternative 4). This indicates that more people are exposed to noise in alternative 4 and that those additionally exposed experience an average hearing loss

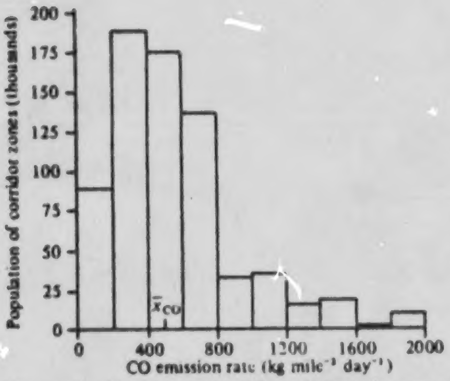

**Figure 5.** Alternative 1: CO emissions for zones in highway corridors.

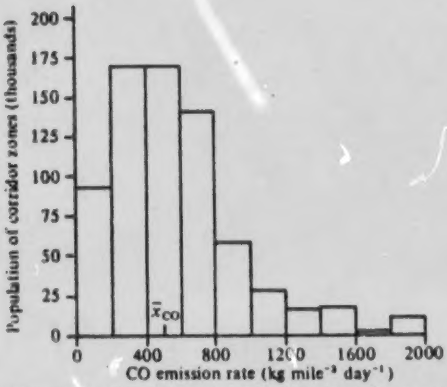

**Figure 6.** Alternative 4: CO emissions for zones in highway corridors.

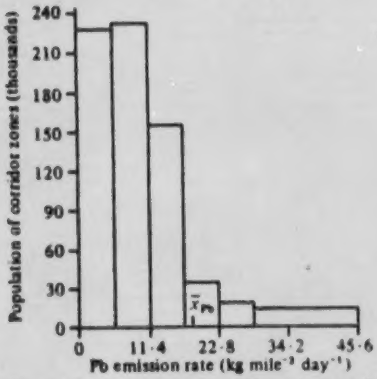

**Figure 7.** Alternative 1: Pb emissions for zones in highway corridors (2·24 g Pb gal$^{-1}$).

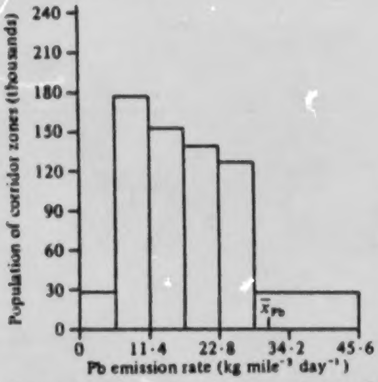

**Figure 8.** Alternative 4: Pb emissions for zones in highway corridors (2·24 g Pb gal$^{-1}$).

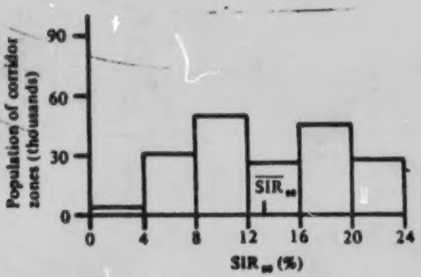

Figure 9. Alternative 1: Speech Impairment Risk for population in highway corridors.

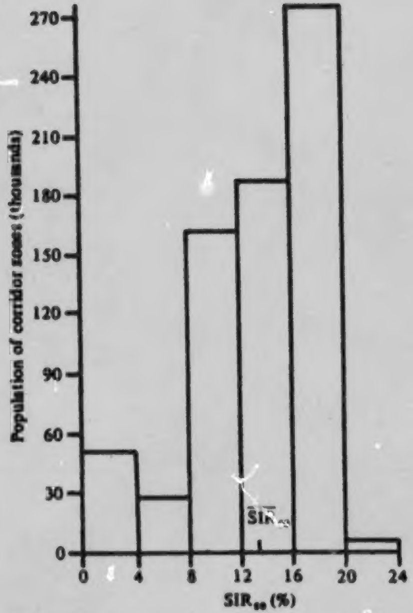

Figure 10. Alternative 4: Speech Impairment Risk for population in highway corridors.

risk similar to those exposed in alternative 1.

## SPECIFIC ZONE ANALYSIS

The basic approach of specific zone analysis was to identify zones with high densities of susceptible populations (persons per square mile) and to compare changes in emission levels in those zones between the base case and the other alternatives. In order to determine what zones were to be considered high density, the total range of density for each population group was identified for the zones in the study area. It was found that for most of the groups the predominant density class was the lowest quartile of their value range. High density zones were, therefore, defined as those zones with densities that exceeded the upper boundary of the first quartile.

Emission levels of each pollutant for these identified high density population zones were measured in a similar manner. The total range of emission densities per square mile for all zones in the study area (regardless of alternative) was divided into six classes. This was done for each of the three pollutants. Focusing only on those zones identified as high density population zones, a consistent scaling technique was developed to identify for each alternative those zones which would be expected to experience a large change in emission levels from the base case. Numbering the emission classes from 1 through 6, all high population density zones with emission levels increasing from the base case two or more classes to class 4 or above were designated high incremental zones. All zones with levels decreasing two or more classes to class 3 or below were designated as low incremental zones. When the number of zones and population size of the low incremental zones were subtracted from those of the high incremental zones, the difference was the net number of "high risk" zones and the size of the population exposed in them. This incremental analysis was performed for each pollutant using the high density zones for populations identified as hypersusceptible to that pollutant. The process was repeated for each of the three alternatives over the base case.

It was found that, as with the overall emissions, the zonal emission levels of CO remained essentially the same for all of the alternatives. The above described incremental analysis could not, therefore, be performed for carbon monoxide. There were, however, 25 zones that had CO emission levels significantly higher than the bulk of the zones evaluated. They were identified as high CO zones, and they were used with the analysis of incremental Pb emission zones to pinpoint those zones which have both high CO and a two-class increase in Pb emissions. As discussed above, high emissions of the two pollutants is a combination for which hypersusceptible populations were identified and enumerated in the study.

Comparison of the high risk zones and populations for each alternative, summarized in Table 4, will enable evaluation of the relative potential health impact of the alternative plans for those specific high risk locations where population densities and emission increases coincide. By locating these zones on a map it can be determined where the potentially most severe impacts may occur, and adjustment of a plan may be made to reduce the risk. A map of high density and high risk zones for long term care facilities is shown in Figure 11.

## DISCUSSION OF RESULTS

The methodology provides plan analysis on two levels. The overall evaluation describes general trends in total potential human pollutant exposure in relation to increases in freeway miles. The specific zone analysis allows identification of those locations where

Table 4. Summary of specific-zone analysis. All comparisons are with respect to alternative 1.

| Population group | Hazard criterion | Population and number of high-risk zones | | | | | |
|---|---|---|---|---|---|---|---|
| | | alternative 2 | | alternative 3 | | alternative 4 | |
| | | population | number of zones | population | number of zones | population | number of zones |
| Total | incremental Pb | 52969 | 9 | 52969 | 9 | 196512 | 27 |
| | high CO | 101421 | 12 | 101421 | 12 | 101421 | 12 |
| | incremental Pb and high CO | 8711 | 1 | 8711 | 1 | 74709 | 9 |
| | incremental $SIR_{50}$ | 66775 | 11 | 66775 | 11 | 233497 | 33 |
| Children 0-2 years old | incremental Pb | 2654 | 7 | 2654 | 7 | 8232 | 18 |
| | high CO | 5458 | 11 | 5458 | 11 | 5458 | 11 |
| | incremental Pb and high CO | 549 | 1 | 549 | 1 | 3646 | 7 |
| | incremental $SIR_{50}$ | 3383 | 11 | 3383 | 11 | 10350 | 25 |
| Day-care centers | incremental Pb | 611 | 8 | 611 | 8 | 1584 | 23 |
| | high CO | 1055 | 12 | 1055 | 12 | 1055 | 12 |
| | incremental Pb and high CO | 163 | 1 | 163 | 1 | 639 | 8 |
| | incremental $SIR_{50}$ | 980 | 18 | 980 | 18 | 2103 | 35 |
| Hospital beds | high CO | 223 | 1 | 223 | 1 | 223 | 1 |
| Long-term care beds | high CO | 551 | 3 | 551 | 3 | 551 | 3 |
| People aged 65 years or older | high CO | 7195 | 9 | 7195 | 9 | 7195 | 9 |
| Elementary-school | incremental Pb | 12101 | 9 | 12101 | 9 | 36502 | 25 |
| | high CO | 16910 | 10 | 16910 | 10 | 16910 | 10 |
| | incremental Pb and high CO | 1840 | 1 | 1840 | 1 | 13464 | 7 |
| | incremental $SIR_{50}$ | 15783 | 10 | 15783 | 10 | 40565 | 29 |
| People below poverty level | incremental Pb | 645 | 1 | 645 | 1 | 4728 | 5 |
| | high CO | 4971 | 5 | 4971 | 5 | 4971 | 5 |
| | incremental Pb and high CO | 645 | 1 | 645 | 1 | 3191 | 3 |

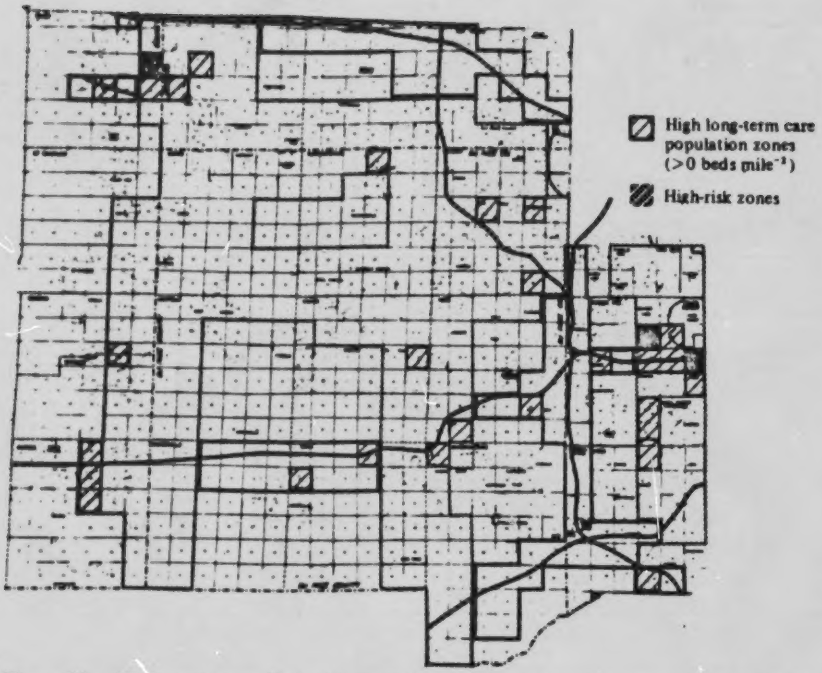

**Figure 11.** All alternatives: High-risk zones for residents of long-term care facilities in highway corridors with respect to carbon monoxide.

concentrations of hypersusceptible populations coincide with large expected increases of the pollutants to which they are susceptible. This analysis summarizes the net high risk zones, taking into account locations where emissions are reduced as well as those where they are increased, thus allowing comparison of the net effects of the various alternatives.

The comparison of the four alternative plans was based on the increments in potential impacts for the three "new freeway" alternatives from the base case of the existing freeway system. Because all the procedures and calculations were used consistently for all the alternatives, valid comparative conclusions as to potential exposures can be made.

The absolute sizes of potentially exposed populations, levels of exposure, and physiological response to exposure could not be obtained. If specific highway location and design details were available, appropriate meteorological diffusion models could be used to calculate air quality at specific locations adjacent to the roadways. When overlayed on population data for those locations, estimates of the numbers exposed and severity of exposure could be made. In order to estimate physiological response to that exposure, quantified dose response relationships based on toxicologic and epidemiologic studies would need to be applied to the exposure estimates.

The relationships which this methodology can provide for evaluating potential health effects of highway systems are:

1. Study area totals of people and pollutants
2. Mapped geographical distributions of amounts of pollutants emitted
3. Mapped distributions of densities of total and hypersusceptible populations

4. Estimates of population-weighted average emission exposure
5. Specific identification of high risk zones with both high emissions and high density populations
6. Location of high risk zones in relation to proposed highway alignments

The advantage of this approach is that it compares not only patterns of emissions, but also the distribution of the people susceptible to them and the potential patterns of exposure. It can be used for any number of pollutants and susceptible populations, and as the results of this case study demonstrate, it can therefore be valuable in characterizing differing trends of potential impacts from each pollutant used in the evaluation. The method provides quantitative and graphical information on potential health impact for use in planning decisions.

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## EVALUATION OF PLANNING ALTERNATIVES:

## ALTERNATIVE FUEL UTILIZATION

Peter A. Scheff

The purpose of the alternative fuel utilization example is to explore a number of health impacts which might result from substituting specific quantities of coal for oil in Chicago. The recent push for the use of more coal resulting from the "Energy Crisis" and our dependence on foreign oil make this type of analysis imperative. As programs are developed to meet these problems, the health costs of each particular energy source must be ascertained if the options are to be rationally assessed.

It should be understood that an incomplete understanding of the effects of pollutants on high-risk populations, along with limitations in the records of occupationally related accidents and illnesses, restrict the scope of this analysis. The purpose of this exercise is to first, generate defensible numbers describing some of the health implications of switching between fossil fuel systems; and second, to develop a methodology that can be applied to different situations and expanded as additional data are made available.

The major components of this example include:

1. The identification and enumeration of hypersusceptible populations.
2. Application of an emission inventory and a validated air pollution dispersion model.
3. Use of dose-response relationships between environmental stress and disease rates.
4. Use of occupational accident and disease rates.
5. The application of a model for predicting the health impacts of energy consumption.

## GROUND RULES

1. The unit of energy used in this example is the "quad." One quad equals a quadrillion Btu's of energy.
2. The analysis is limited to the fossil fuel cycles which produce electrical power for industry, space heating, and other consumer uses.
3. The health effects considered are accidents associated with fossil fuel cycles, morbidity due to occupational exposure, and morbidity in high-risk populations in Chicago exposed to air pollutants from fossil fuel combustion. The effects of water pollution and industrial pollution sources not directly associated with the production of electric energy are not considered.
4. A diffusion model and emission inventory derived from studies carried out by the State of Illinois and Argonne National Laboratory are used to define the relationship between energy input and air quality.
5. The only air pollutants considered are sulfur dioxide and suspended sulfates. Particulates, nitrogen oxides, trace metals, and other air pollutants and pollutant interactions are not considered.

## DESCRIPTION OF HEALTH-ENERGY MODEL

Figure 1 is a graphic representation of a model for predicting the health impact of energy consumption. To apply this model, the relationship between energy input to the Chicago Metropolitan Region and air quality must be defined. This is done by first defining the total amount of energy consumed in terms of an amount of coal, oil and natural gas. The emissions which form this specific energy mix can then be estimated. Changing the relative amounts of fossil fuels results in changing emission quantities. Since air quality is a function of emissions, a validated air pollution model can predict changes in air quality resulting from changes in the relative amounts

Model for Quantitating and predicting Health Impact of Energy Consumption

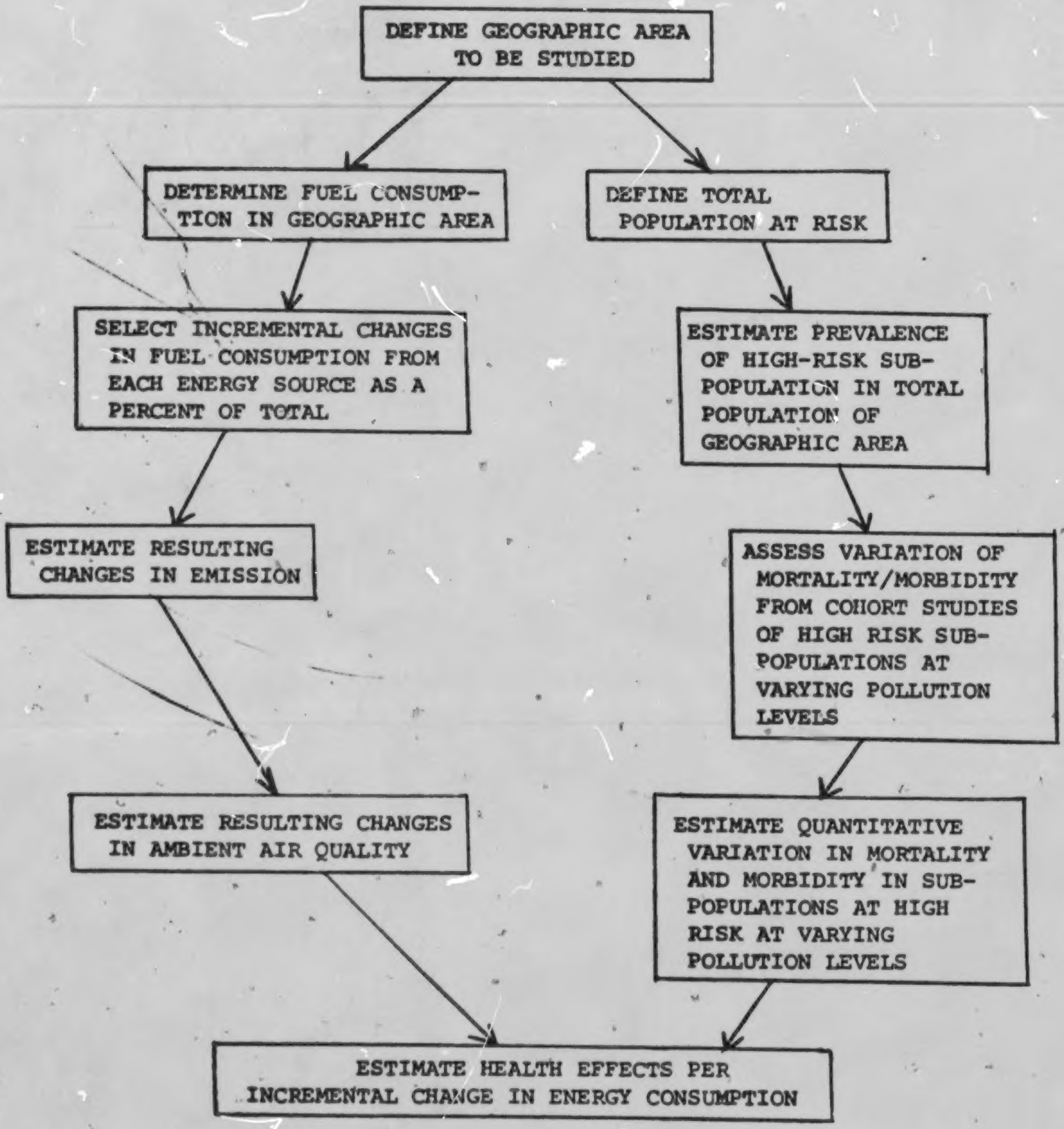

DEFINE GEOGRAPHIC AREA TO BE STUDIED
DETERMINE FUEL CONSUMPTION IN GEOGRAPHIC AREA
DEFINE TOTAL POPULATION AT RISK
SELECT INCREMENTAL CHANGES IN FUEL CONSUMPTION FROM EACH ENERGY SOURCE AS A PERCENT OF TOTAL
ESTIMATE PREVALENCE OF HIGH-RISK SUB-POPULATION IN TOTAL POPULATION OF GEOGRAPHIC AREA
ESTIMATE RESULTING CHANGES IN EMISSION
ASSESS VARIATION OF MORTALITY/MORBIDITY FROM COHORT STUDIES OF HIGH RISK SUB-POPULATIONS AT VARYING POLLUTION LEVELS
ESTIMATE RESULTING CHANGES IN AMBIENT AIR QUALITY
ESTIMATE QUANTITATIVE VARIATION IN MORTALITY AND MORBIDITY IN SUB-POPULATIONS AT HIGH RISK AT VARYING POLLUTION LEVELS
ESTIMATE HEALTH EFFECTS PER INCREMENTAL CHANGE IN ENERGY CONSUMPTION

Figure 1.

of fuel consumed.

The other side of the model defines relations between air quality and human health. This is accomplished by first defining the total population at risk and then by identifying and quantitating high-risk subpopulations within the general population. Epidemiologic cohort studies of these high-risk groups are used to define variations in morbidity and mortality as a function of air quality. These relationships are then extrapolated to the total high-risk subpopulations defined in the study area. When combined with the air quality model, incremental changes in health resulting from changes in fuel input can be predicted.

Occupational accidents from production, processing, and transportation are incorporated into the model by first defining accident rates per unit of energy produced. Then, by applying these rates to the specific changes in energy consumption defined above, net changes in the number of accidents for each energy cycle are calculated. To complete the application of the model, occupational disease rates associated with the energy cycles being assessed above are identified. As with the occupational accident data, the occupational disease rates are applied to the changes in fuel consumption to calculate the net changes in the amount of disease associated with the change of energy consumption.

## RELATIONSHIP BETWEEN AIR QUALITY AND ENERGY USE

Energy produced by combustion of fossil fuels can be related to accident and occupational health data by consideration of the total amounts of coal, oil and gas used. The determination of air quality, due to these combustion processes, allows evaluation of the air quality caused by different usages of coal, oil and gas. By using different values of air quality, estimations can be made for certain disease categories of the number of environmentally associated occurrences related to different fractions in each fuel category. The air pollution model then supplies the necessary linkage between environmental disease and fuel production and use.

The information relating fossil fuel use to air quality for the City of Chicago is derived from studies carried out by the State of Illinois and Argonne National Laboratory (2-4,10,14). These studies formed the basis for the Illinois Air Emission Implementation Plan (10). This model is used because it has been partially validated with respect to measurements of air quality in the Chicago area. In order to calculate air quality within the City of Chicago it is necessary to include emissions from pollution sources both within the City, and those outside the City which affect air quality in Chicago.

Three situations of different energy usage for the Chicago Metropolitan Region are proposed and a relationship developed between the fraction of energy used as coal, the fraction of energy used as oil, and the resulting annual geometric mean concentration for sulfur dioxide. The basic assumptions underlying this relationship are as follows:

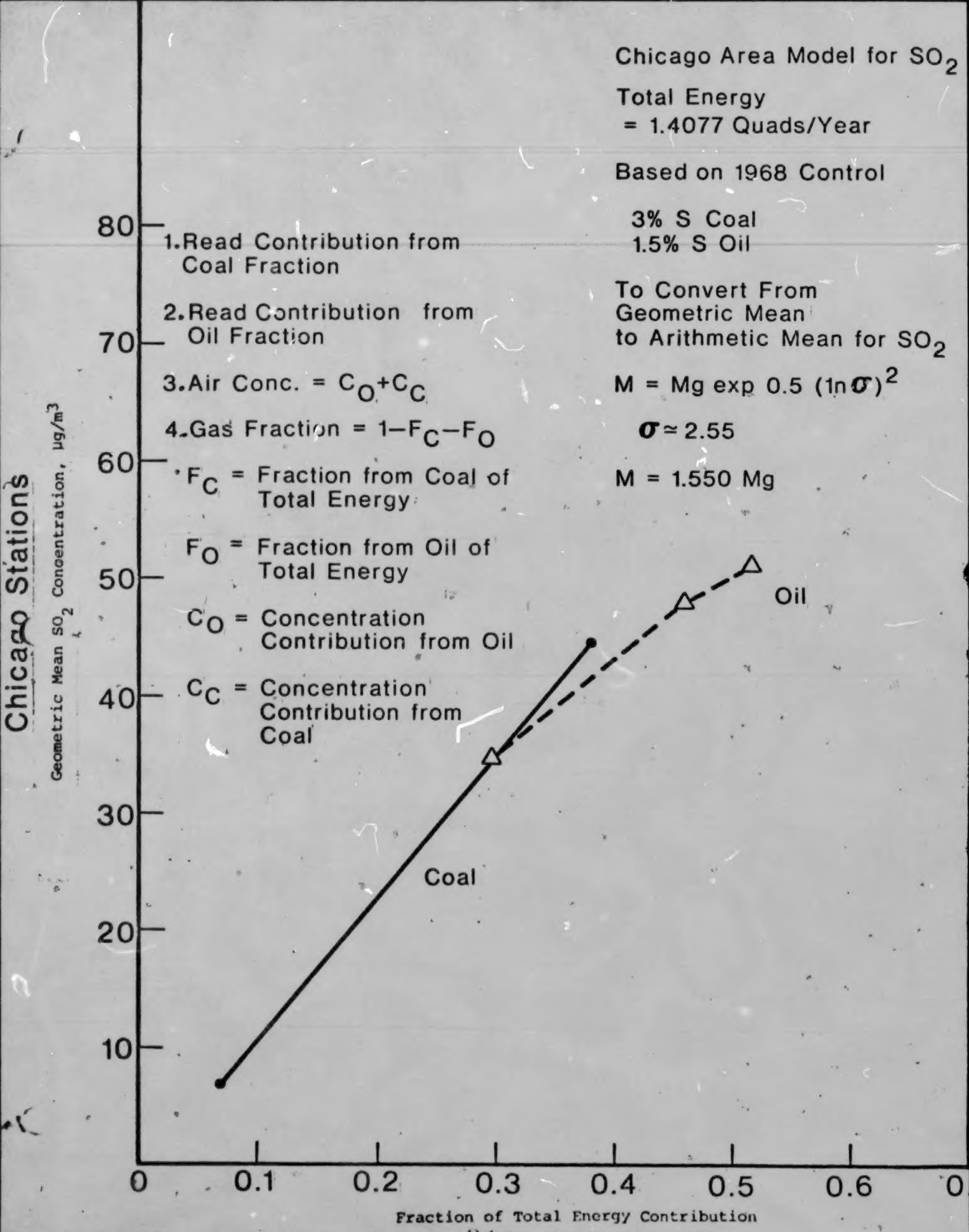
1. The total energy usage for the Chicago Major Metropolitan area is estimated at 1.408 quads per year.
2. The emissions of particulate matter and sulfur dioxide are based on 1968 control situations in the Chicago region.
3. The sulfur content for coal and oil is assumed to be 3% and 1.5%, respectively.
4. The heating value of coal is taken as 11,000 Btu/lb and the heating value for oil is taken as 148,000 Btu/gal.
5. The combustion of natural gas is assumed not to add any particulate or $SO_2$ to the atmosphere.

Using the emission inventory as input information for the diffusion model it is possible to determine the amounts of coal, oil and gas used for three different cases. The diffusion model calculates air concentration in terms of an annual geometric mean. These concentrations are available for the whole Chicago Major Metropolitan area. However, the geometric mean concentration is different at different locations in the region. Therefore, an area weighted geometric mean for the City of Chicago is determined for each of the three cases. The geometric mean for sulfur dioxide is then related to the amounts of coal and oil used in each case. This is done by ratioing the air quality (or air concentration) for $SO_2$ on the basis of relative amounts of coal and oil utilized. The results of this estimation procedure are shown in Figure 2. This gives the relationship between the fraction of total energy from each fossil fuel and its contribution to the annual geometric mean for sulfur dioxide. To calculate the total air concentration one reads the contribution from the coal fraction and the contribution from the oil fraction, and adds these two together. This total will be equal to the annual geometric mean. The gas fraction will be one minus the coal fraction plus the oil fraction. The same kind of procedure can be carried out for other fuel usages and for other cities, if a validated diffusion model exists for these urban areas.

## RELATIONSHIPS BETWEEN ENVIRONMENTAL HEALTH AND AIR QUALITY

### Identification of Hypersusceptible Populations

The purpose of this section is to propose a set of relationships between environmental quality and human health to which the predicted changes in air quality can be applied. The first step in determining these relationships is to identify specific subgroups within the general population which are at risk to the pollutants being assessed. For this example, the following three groups are used: 1) Children five years old or under; 2) people with asthma; and 3) males, ages



Chicago Area Model for $SO_2$

Total Energy
= 1.4077 Quads/Year

Based on 1968 Control

3% S Coal
1.5% S Oil

To Convert From Geometric Mean to Arithmetic Mean for $SO_2$

$M = Mg \exp 0.5 (\ln \sigma)^2$

$\sigma \simeq 2.55$

$M = 1.550\ Mg$

1. Read Contribution from Coal Fraction
2. Read Contribution from Oil Fraction
3. Air Conc. = $C_O + C_C$
4. Gas Fraction = $1 - F_C - F_O$

$F_C$ = Fraction from Coal of Total Energy

$F_O$ = Fraction from Oil of Total Energy

$C_O$ = Concentration Contribution from Oil

$C_C$ = Concentration Contribution from Coal

55 and older, with chronic bronchitis grades 3 and 4.

## Dose Response Relationships

Figure 3 shows the relation between annual arithmetic averages of sulfur dioxide in the air and number of acute respiratory illness expected per person per year for preschool children and nursery school children (9,11). This figure is derived from the New York and Chicago studies of acute respiratory disease from the CHESS program. These two studies are chosen because the data were collected, analyzed, and presented in a similar fasion, and the pollution mix of the two cities is fairly comparable. The illness rate includes both upper and lower respiratory disease. An upper respiratory disease is defined as a cough, head cold, sore throat, sinus or postnasal drip, or runny nose. A lower respiratory disease is defined as a chest cold, deep productive cough, wheezing, croup, bronchitis, or pneumonia. Children are chosen in this study because they have a much higher incidence of acute respiratory disease than the population at large, and they are not biased by personal factors such as smoking. The New York study lasted for 30 weeks and the Chicago study lasted for 46 weeks. Both studies were run during the influenza season. In Figure 3 the rates from both studies are adjusted to extend over a period of one year. Because the studies were made during the seasons with the highest illness rate, the extrapolation of the rates to one year may be a little high.

Figure 4 shows a relation between average daily concentration of suspended sulfate and asthma attack rate per person per day for asthmatics of all ages (8). This figure is derived from the New York CHESS study of asthmatics and is chosen because the environmental pollutants of New York are more similar to those of Chicago than the other cities studied. Because of the importance of the effect of temperature on asthma attack rates, two relationships are identified.

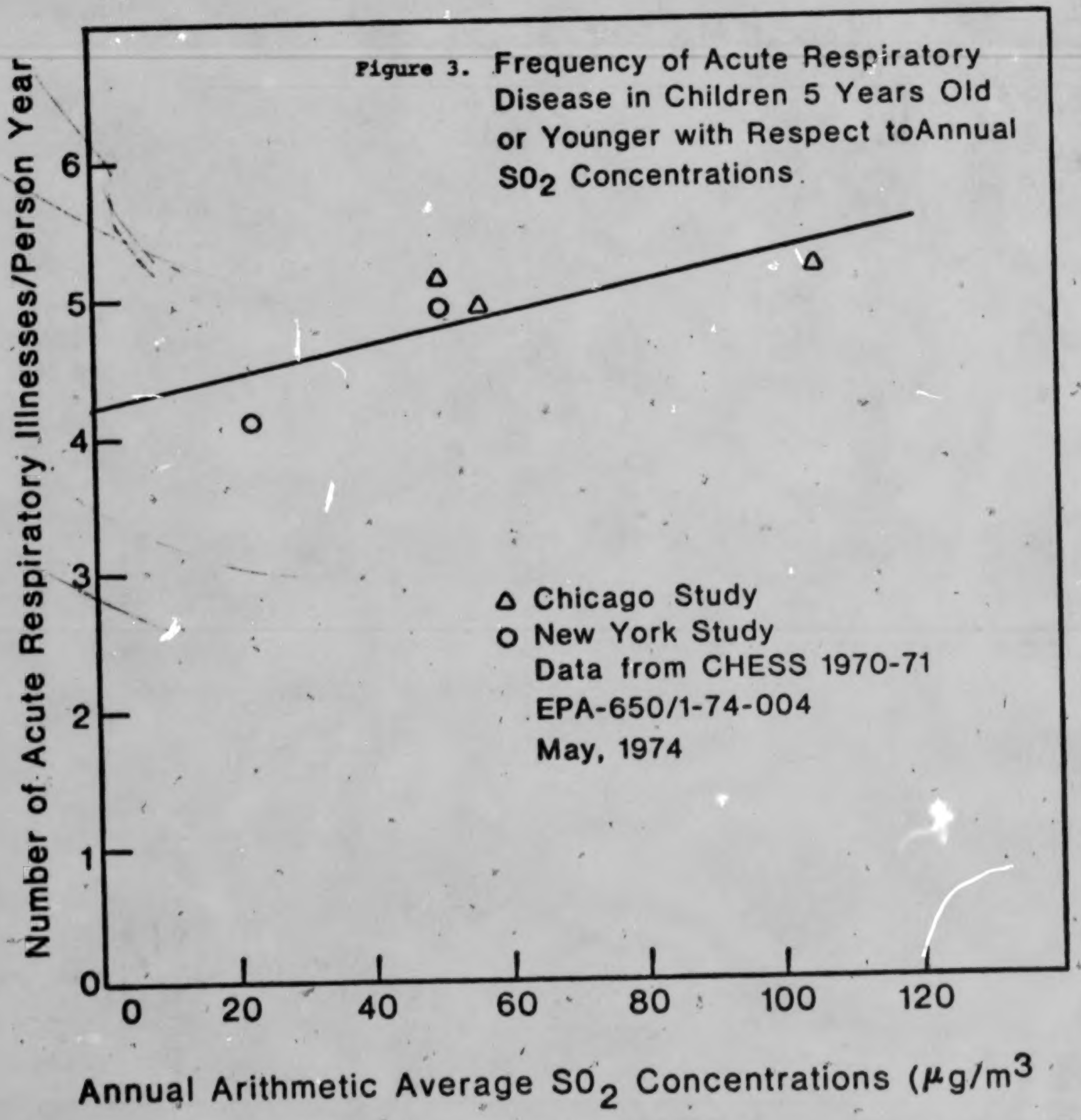
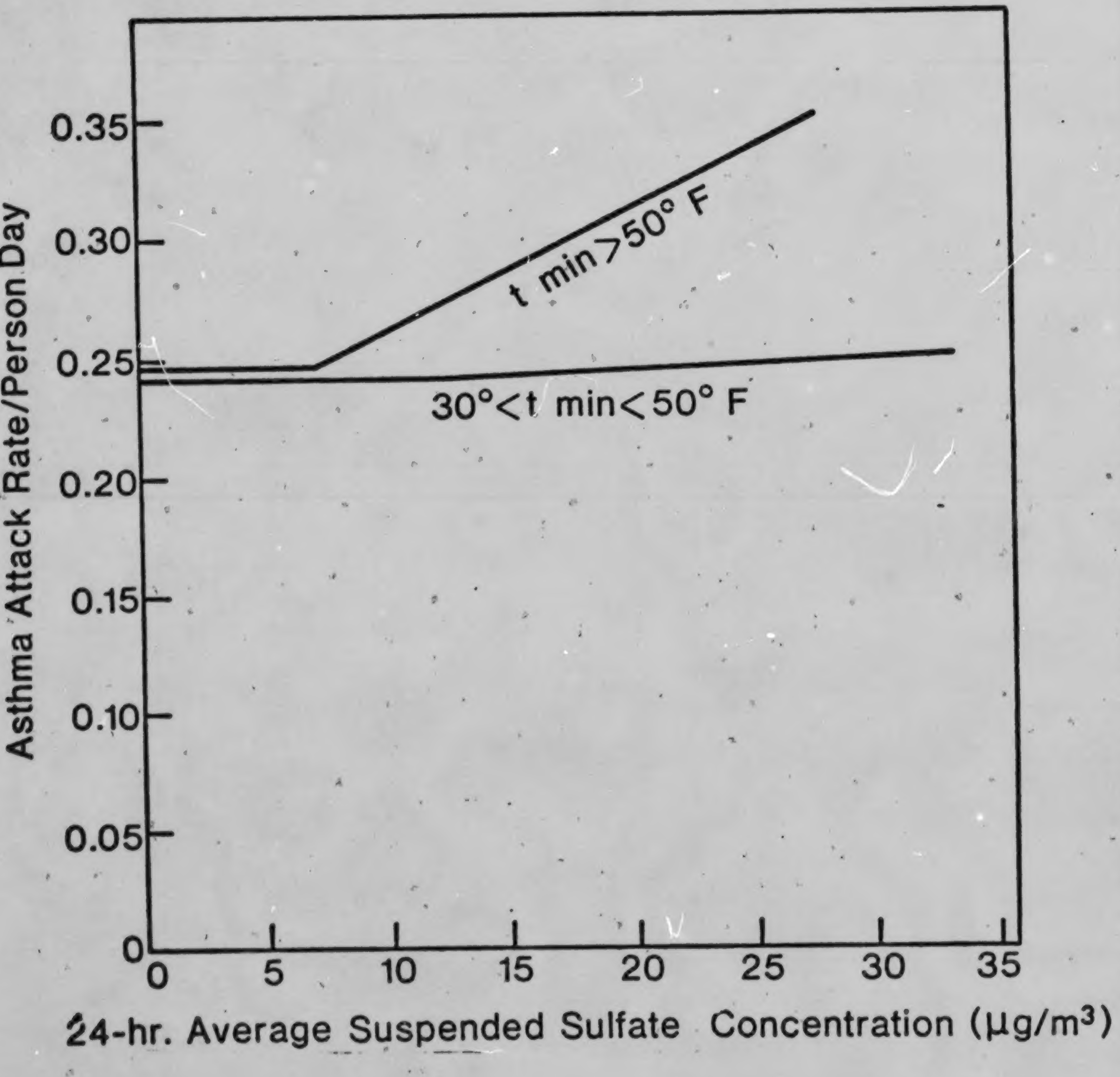


**Figure 3.** Frequency of Acute Respiratory Disease in Children 5 Years Old or Younger with Respect to Annual $SO_2$ Concentrations.

Data from CHESS 1970-71
EPA-650/1-74-004
May, 1974

Figure 4. Asthma attack rate versus suspended sulfate concentration.



One holds when the minimum daily temperature is greater than 50°F, and the other is valid when the minimum daily temperature is between 30 and 50°F. Ambient pollution does not appear to have a measurable effect on the asthma attack rate when the minimum daily temperature is below 30°F.

Figure 5 shows a relation between daily average $SO_2$ levels and expected number of sick days per day exposed for males aged 55 and over with chronic bronchitis grades 3 and 4 (5). This figure is derived by dividing the number of sick days at pollution concentration i per person by number of days exposed at level i per person. A chest illness or sick day is defined as one on which a patient was bothered much more than usual by his chest for most of the day, had more cough than usual because of his chest, or had a chest cold. Since the relationship shown in Figure 5 represents data collected from October 1 to November 30, 1967, extrapolation to an entire year may introduce some inaccuracy.

## Enumeration of Hypersusceptible Populations

In order to apply these relationships to the energy health model, an estimate of the number of people at risk is needed. The number of children five years old or younger at risk in Chicago is taken from the 1970 census (12):

number of children = 343,100

The number of people with asthma at risk in Chicago is calculated as follows (12,15):

number of asthmatics = population of Chicago
x asthma prevalence rate
= 3,366,957 x 0.03
= 101,009

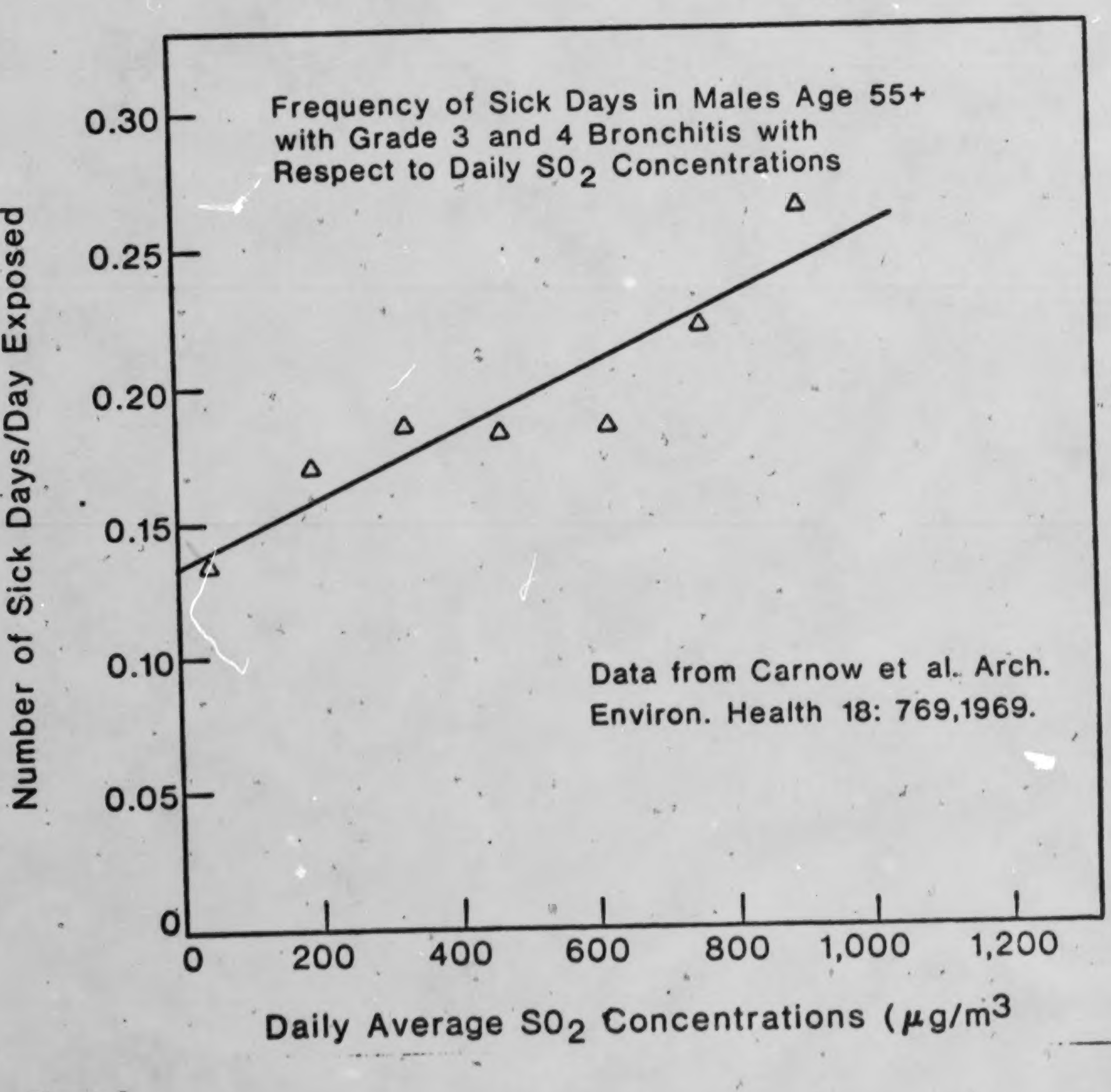

Frequency of Sick Days in Males Age 55+ with Grade 3 and 4 Bronchitis with Respect to Daily $SO_2$ Concentrations
Number of Sick Days/Day Exposed
0.30
0.25
0.20
0.15
0.10
0.05
0
0
200
400
600
800
1,000
1,200
Daily Average $SO_2$ Concentrations ($\mu g/m^3$
Data from Carnow et al. Arch. Environ. Health 18: 769,1969.

Figure 5.

The number of males age 55 and older with chronic bronchitis grades 3 and 4 at risk in Chicago is calculated as follows (12,16,17):

number of bronchitics = numbers of males ages 55+ x bronchitis prevalence rate x bronchitis severity rate

= 307,309 x 0.0285 x 0.179

= 1,567

## APPLICATION OF ENERGY-HEALTH EFFECTS MODEL: CHICAGO EXAMPLE

### Energy and Environmental Quality

The energy input into the Chicago Metropolitan Region is estimated to be 1.407 quads per year. For the Base Case, 0.234 quads of coal at 3% sulfur, 0.714 quads of oil at 1.5% sulfur and 0.459 quads of gas have been chosen. This corresponds to 17% of the total energy from coal, 51% from oil, and 32% from gas. From the air quality model it is determined that the Base Case energy input results in an annual geometric mean of 69.4 $\mu g/m^3$ $SO_2$. The model is applied to three specific changes in energy consumption: Case I - change 0.1 quad from oil to coal; Case II - change 0.2 quad from oil to coal; and Case III - change 0.3 quad from oil to coal. For Case I, this results in an annual geometric mean concentration of 74.1 $\mu g/m^3$ $SO_2$, an incremental increase of 4.7 $\mu g/m^3$. The incremental increase for Case II is 7.1 $\mu g/m^3$ and for Case III is 9.3 $\mu g/m^3$. Annual arithmetic means are calculated using equation 1 and a standard deviation ($\sigma$) of 2.55 for $SO_2$. This value of $\sigma$ is based on 1968 Chicago air quality data (13).

$$m = M_g \exp \left[ 0.5 \ (\ln \sigma)^2 \right] \qquad (1)$$

$m$ = arithmetic mean

$M_g$ = geometric mean

$\sigma$ = standard deviation

Annual arithmetic sulfate ($SO_4$) concentrations are calculated from equation 2 (1).

$$C_{SO_4} = C_{SO_2} / (0.0475\, C_{SO_2} + 1.69) \qquad (2)$$

$C_{SO_4}$ = annual arithmetic $SO_4$ concentration

$C_{SO_2}$ = annual arithmetic $SO_2$ concentration

Geometric mean $SO_4$ concentrations are calculated from equation 1. The standard deviation for $SO_4$ is 1.44 (7).

A complete list of annual arithmetic and geometric mean $SO_2$ and $SO_4$ concentrations, along with the incremental changes between the Base Case and Cases I, II, and III are shown in Table 1.

## Occupational Accident and Disease Analysis

Rates per quad for fatal and nonfatal accidents for the petroleum and coal industry and for the occupational disease coal worker's pneumonconiosis are used in this section. These rates can be applied to the incremental changes in energy use for the three cases. The net change in occupational accidents and disease associated with each case can then be calculated. For example, the fatal accident rate for the coal industry is 22.4 deaths/quad, and for the oil and gas industry is 2.5 deaths/quad. Case II shows that there is an increase of 0.2 quads from coal which results in an extra 4.48 accidental deaths. However, there is also a decrease of 0.2 quads from oil which results in a decrease of 0.5 accidental deaths. Thus the net change in accidental deaths due to the change of 0.2 quads from oil to coal is 4.48 - 0.50 = 3.98 added accidental deaths. In exactly the same way a net increase of 85.8 accidental injuries associated with Case II

Table 1. Energy Use and Air Quality Summary for Chicago Example, Constant Energy Input, 1968

| Energy Use (Quads) | Base Case | Case I | Case I - Base Case | Case II | Case II - Base Case | Case III | Case III - Base Case |
|---|---|---|---|---|---|---|---|
| Coal | 0.234 | 0.334 | +0.100 | 0.434 | +0.200 | 0.534 | +0.300 |
| Oil | 0.714 | 0.614 | -0.100 | 0.514 | -0.200 | 0.414 | -0.300 |
| Gas | 0.459 | 0.459 | - | 0.459 | - | 0.459 | - |
| Total | 1.407 | 1.407 | - | 1.407 | - | 1.407 | - |
| **Air Quality** ($\mu g/m^3$) | | | | | | | |
| $SO_2$ | | | | | | | |
| Geometric Mean | 69.4 | 74.1 | +4.7 | 76.5 | + 7.1 | 78.7 | + 9.3 |
| Arithmetic Mean | 107.5 | 114.8 | +7.3 | 118.6 | +11.1 | 122.0 | +14.5 |
| $SO_4$ | | | | | | | |
| Geometric Mean | 14.79 | 15.05 | +0.26 | 15.16 | + 0.37 | 15.25 | + 0.46 |
| Arithmetic Mean | 15.81 | 16.08 | +0.27 | 16.20 | + 0.39 | 16.30 | + 0.49 |

is calculated. There is also an increase of 86.6 cases of coal worker's pneumonconiosis associated with the increase of 0.2 quads of coal for Case II. The complete occupational results for Cases I, II, and III are presented in Table 2.

## Environmental Health Analysis

The environmental health analysis incorporates information derived in the air quality analysis section and environmental health data section. The question being answered here is what are the health effects of the incremental changes of fuel consumption between the Base Case and Cases I, II, and III. In Case III, for example, the incremental increase in the annual arithmetic mean $SO_2$ concentration is 14.5 $\mu g/m^3$ over the value of the Base Case. Figure 3, the frequency of acute respiratory disease in children, relates the number of illnesses expected per person per year to an ambient $SO_2$ annual arithmetic mean. Applying the Case III increase of 14.5 $\mu g/m^3$ to this relationship results in an incremental increase of 0.163 illnesses per person per year. Multiplying this increase by the total population at risk predicts a total increase of 55,943 excess illnesses per year of children five years old or younger in the City of Chicago. By applying the increases in the ambient arithmetic mean concentration of $SO_2$ for Cases I and II in the same fashion as above, increases per year of 28,164 illnesses and 42,825 illnesses, respectively, are calculated.

Estimating the excess number of asthma attacks associated with an incremental increase in the ambient level of $SO_2$ is a more complex task. Figure 4 relates a daily asthma attack rate to the average daily concentration of suspended sulfates for two temperature ranges. However, the arithmetic mean sulfate data from Table 1 is reported in terms of annual averages. In order to calculate the increase in the number of asthma attacks, the expected number of days at every possible sulfate concentration for the year associated with each annual

Table 2. Accident, Occupational and Environmental Disease Summary for Chicago Example, 1968

| Accidents | Base Case | Case I | Case I - Base Case | Case II | Case II - Base Case | Case III | Case III- Base Case |
|---|---|---|---|---|---|---|---|
| Fatal Coal | | | + 2.24 | | + 4.48 | | + 6.72 |
| Fatal Oil & Gas | | | - 0.25 | | - 0.50 | | - 0.75 |
| Net Fatal Accidents | | | + 1.99 | | + 3.98 | | 5.97 |
| Non Fatal Coal | | | +65.5 | | +139.00 | | +208.5 |
| Non Fatal Oil & Gas | | | -26.6 | | - 53.2 | | - 79.8 |
| Net Non Fatal Accidents | | | +38.9 | | + 85.8 | | +128.7 |
| **Occupational Diseases** | | | | | | | |
| Pneumoconiosis | | | | | | | |
| Diagnosed | | | +21.2 | | + 42.4 | | + 63.6 |
| Suspected | | | +22.1 | | + 44.2 | | + 66.3 |
| Total Pneumoniosis | | | +43.3 | | + 86.6 | | +129.9 |
| **Environmental Diseases** | | | | | | | |
| Childhood Respiratory Disease | | | +28,164 | | +42,825 | | +55,943 |
| Asthma Attacks | 7,351,132 | 7,375,778 | +24,646 | 7,381,031 | +29,899 | 7,388,404 | +37,272 |
| Days of Illness for Males 55+,Bronchitics Grade 3 & 4 | 84,367 | 84,931 | + 564 | 85,198 | + 831 | 85,449 | + 1,082 |

geometric mean concentration of the four cases must be determined. Because daily sulfate concentrations appear to be log-normally distributed over a year (7) the percent of time during the year expected at different concentrations can be calculated. The probability that a daily pollution level (assuming the pollutant is log-normally distributed) will be less than any specified concentration is given by equation 3.

$$P = \int_{-\infty}^{X'} (2\pi)^{-\frac{1}{2}} \exp \left[ - 0.5 X^2 \right] dX \qquad (3)$$

$X = \ln (c/m_g)/\ln\sigma$

$C$ = concentration

$P$ = probability that a particular daily pollution level is $\leq c'$

$X'$ = value of X at concentration $c'$

$c'$ = specified concentration

$m_g$ = geometric mean

$\sigma$ = standard deviation

The probability that the mean daily pollution level will be within a specific concentration range $c_1$ to $c_2$ equals the difference between the probability that the daily pollution level will be below $c_2$ and the probability that the daily pollution level will be below $c_1$.

Using equation 3, the probability of the daily mean $SO_4$ concentration falling within the following ranges can be calculated for each annual geometric mean concentrations listed in Table 1: 0-7.3 $\mu g'/m^3$, 7.3-10, 10-11.9, 11.9-15,15-20,20-25, 25-30, 30-35, and >35 $\mu g/m^3$.

In order to translate this information into the number of days expected for each concentration range for each of the 2 curves, the number of days during which each relationship applies must be determined. Meteorological data from the City of Chicago for 1968 identify 147 days where the minimum daily temperature ($T_{min}$) of Chicago was $\geq 50^{\circ}$ F, and 126 days where the minimum daily temperature was between 30 and 50° F. The sulfate concentration is assumed to be log-normally distributed for both temperature ranges. The number of days expected at each sulfate concentration is calculated by multiplying the probability for each concentration by the number of days at each temperature level. Using equation 4 the number of attacks expected for each concentration range for both curves are summed together to get the total number of asthma attacks expected per year for each of the four annual geometric mean concentrations of $SO_4$.

$$A = P_A \sum_{i=o} (D_{i>50})(R_{i>50}) + \sum_{i=o} (D_{i>30})(R_{i>30}) \tag{4}$$

$A$ = number of asthma attacks per year

$P_A$ = number of asthmatics in Chicago

$D_{i>50}$ = number of days at concentration i for $T_{min} > 50°$ F

$R_{i>50}$ = asthma attack rate per person day for $T_{min} > 50°$ F

$D_{i>30}$ = number of days at concentration i for $30°$ F $< T_{min} < 50°$ F

$R_{i>30}$ = asthma attack rate per person day for $30°$ F $< T_{min} < 50°$ F at concentration i

This procedure estimates that the Base Case annual geometric mean sulfate concentration of 14.79 $\mu g/m^3$ is accompanied with 7,351,132 asthma attacks, Case I by 7,375,778 attacks, Case II by 7,381,031 attacks and Case III by 7,388,404. Subtracting the Base Case from Case I results in an incremental increase of 24,646 asthma attacks associated with the transfer of 0.1 quad from oil to coal for the City of Chicago (Case I). For Case II the increase is 29,899 and 37,272 attacks would be predicted for Case III (see Table 2).

The final component of the energy health example, the frequency of sick days of male bronchitics, is very similar to the asthma example. The $SO_2$ concentrations in Figure 5 are reported as daily averages rather than annual averages. As with the asthma example, the percent of time expected at every possible $SO_2$ concentration can be calculated. Extrapolating the relation to one year, the number of days expected at 9 concentration ranges for the four cases is estimated. Using equation 5 the total number of sick days expected per year for each case can be calculated:

$$S = P_B \sum_{i=o} (D_i)(R_i) \qquad (5)$$

$S$ = number of sick days per year
$P_B$ = number of male bronchitics age 55+, with severity grades 3 & 4 in Chicago
$D_i$ = number of days at concentration i

$R_i$ = rate of sick days per person day exposed at concentration i

The results of these summations are listed in Table 2. As with the asthma example, differences are calculated to determine the incremental increase in the number of sick days expected with each case. 564 excess sick days are associated with Case I, 831 with Case II and 1,082 with Case III.

## CONCLUSION

Most of the Environmental measurements and calculations used in this report are based on 1968 conditions. The health data, defining childhood disease and asthma attack rates, were collected in the early 1970's. The bronchitis data is even older. The conclusions of this example must be interpreted carefully knowing that environmental conditions have changed. These limitations, however, do not invalidate the health impacts listed in Table 2. This table is derived from real data and represents a real risk if similar fuel switches to the ones hypothesized in this report were enacted today. In addition, this example develops the procedures necessary to predict the health costs of energy use. As new occupational and environmental health data become available, as well as improved environmental quality data, they can be incorporated into this analysis and its conclusions updated (6).

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# EVALUATION OF PLANNING ALTERNATIVES: SHOPPING CENTER ANALYSIS

R. G. Hankett

## INTRODUCTION

Many commonplace activities of urban life produce concomitant effects which impact public health. Although an individual shopping trip may seem a relatively innocuous activity, many such trips to a central location, such as a regional shopping center, can have significant effect upon local air quality. In particular, the additional traffic increases the emission of pollutants such as carbon monoxide (CO), lead (Pb), particulate materials (PM), and particulate sulfates ($SO_4$). Each of these is potentially hazardous to human health.

Vehicular traffic patterns are characterized by diurnal and seasonal volume variations. Local meteorological conditions also vary so that pollutants are either trapped or dispersed by the atmosphere. The simultaneous occurrence of high traffic volumes, such as during holiday periods, and adverse meteorology, such as inversions, is not uncommon. As a result, air pollutants accumulate in short-term cyclic or episodal patterns. Of special concern in this regard are hypersusceptible groups such as the elderly, the very young and persons with lung and heart diseases. Such individuals may suffer adverse effects from even short-term exposures to high levels of contamination. Thus, in the consideration of any activity which involves significant increases in automobile traffic, it is essential to evaluate the potential effects of short-term variation in air quality upon hypersusceptibles.

In this case study (5), it is proposed to construct a regional shopping center in the Central Business District (CBD) of a metropolitan area suburb. The center would incorporate a 2,000 car parking garage and be served by an existing arterial street network and rapid transit system. Detailed traffic network assignments are available based upon "design day" trip volumes and modal splits. Since the CBD is surrounded by a mixture of residential, institutional and community service land uses, a detailed evaluation of potential air quality effects is desired. Baseline existing air quality data are not available. Figures 1 and 2 show the street network of the suburban CBD before and after implementation of the proposed shopping center. Design traffic volumes are also indicated.

The task of evaluating the air quality effects and potential health risks of the proposed undertaking is approached as a four-step process:

a) Identify and quantify pollutant emission sources.
b) Analyze changes in ambient air quality.
c) Identify hypersusceptible receptor groups exposed to increased contamination.
d) Evaluate severity of health-based impacts upon receptors.

## AIR POLLUTANT EMISSION SOURCES

The primary air pollutant emission source associated with shopping centers is the automobile, traveling over the roadway network serving the center. Air pollutant emissions produced by a vehicular transportation facility or network are a function of the vehicle population, average speeds, idling times, operating characteristics and travel patterns. The factors of vehicle population, speed, idling and operating mode may be accounted for via a composite emission factor for the pollutant of interest. Travel patterns may be represented in terms of vehicle miles traveled (VMT) over a given highway network or within a given area. For the purposes of this study, the composite emission factor calculation methods presented in Supplement 5 (Dec. 1975) to U. S. Environmental Protection Agency Publication AP-42 have been utilized (11,12). Data for vehicular travel was generated from roadway network traffic assignments by basic traffic

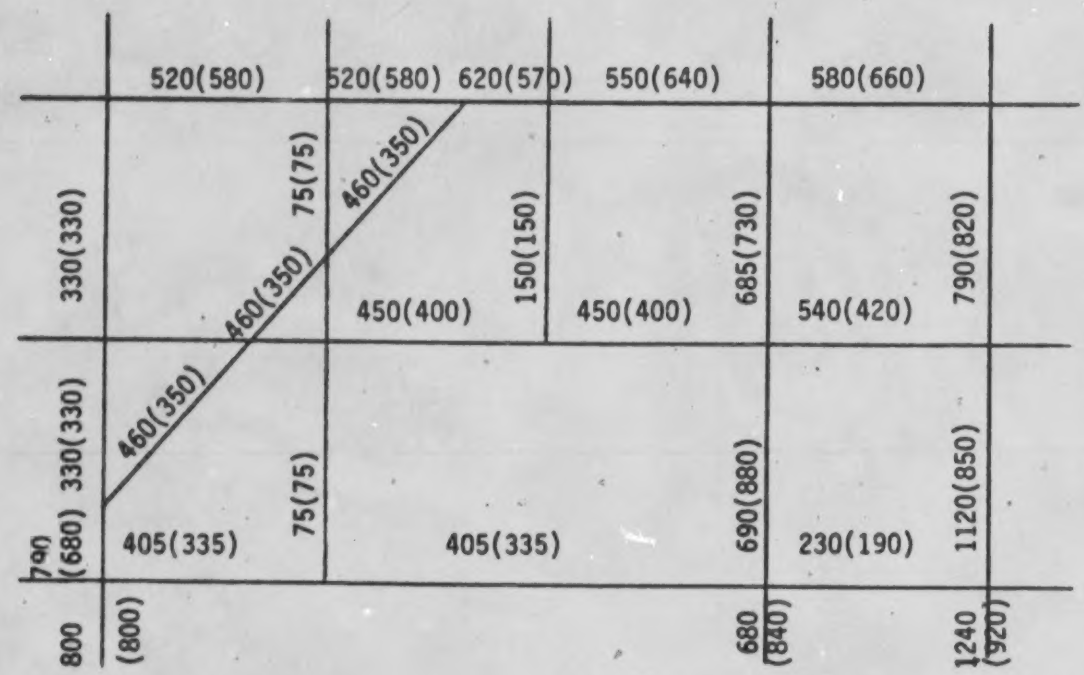

Figure 1. Existing street network-peak hour traffic volumes are indicated on each segment--weekday (Saturday), vehicles/hr.

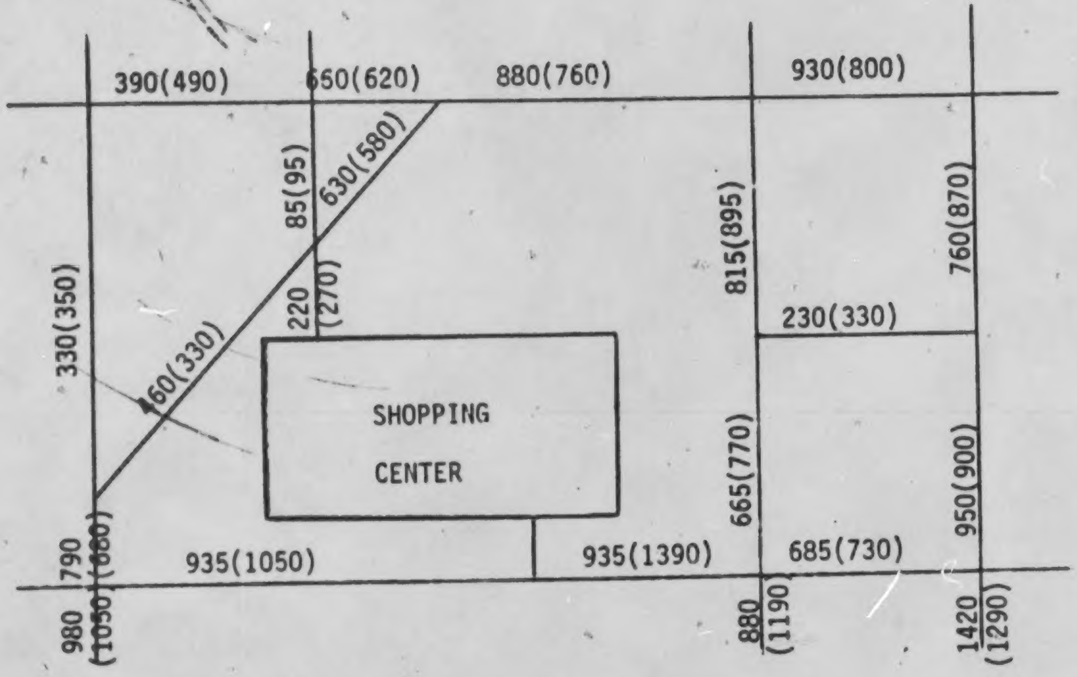

Figure 2. Street network with proposed shopping center--peak hour traffic volumes are indicated on each segment--weekday (Saturday), vehicles/hr.

analysis techniques.

Carbon monoxide (CO), lead (Pb), particulates (PM) and particulate sulfates ($SO_4$) have been selected as pollutants of interest for study. Each of these materials is emitted in significant quantities by motor vehicles and each is potentially hazardous to human health.

## EMISSION FACTORS

To develop an emission factor, it is necessary to consider the following questions:

1). What calendar year is the calculation for?

2). What is the average speed for the area of concern?

3). What is the vehicle mix in terms of types, age and annual travel?

4). What is the ambient temperature?

5). What is the hot start/cold start operational ratio?

In this case study, the appropriate responses are:

1). 1978 is the estimated year of project completion and is, therefore, used for both the proposed and "do-nothing" cases.

2). Traffic on the CBD arterial streets is estimated to travel at an average speed of 15 miles per hour.

3). The vehicle age and mileage mix used is shown in the table below (6). A population of 100% light-duty vehicles (autos) is assumed since the project is located in a CBD area.

| AGE | FRACTION OF TRAVEL | YEAR |
|---|---|---|
| 0 | 0.102 | 1978 |
| 1 | 0.159 | 1977 |
| 2 | 0.172 | 1976 |
| 3 | 0.130 | 1975 |
| 4 | 0.097 | 1974 |
| 5 | 0.083 | 1973 |
| 6 | 0.073 | 1972 |
| 7 | 0.058 | 1971 |
| 8 | 0.042 | 1970 |
| 9 | 0.031 | 1969 |
| 10 | 0.020 | 1968 |
| 11 | 0.011 | 1967 |
| 12 and over | 0.015 | pre- 1967 |

In this vehicle mix, autos from 1975-1978 are considered to be equipped with catalytic converters, while the others are non-catalytic.

4). An average temperature of 65° F is assumed, since air pollution episodes occur most frequently during fall seasons.

5). For worst case conditions such as "rush-hours" or peak shopping periods (1-8 hrs), it is assumed that the entire vehicle population is in either a hot or cold-start operating mode. For longer periods such as daily averages, the hot/cold start operational characteristics typical of a metropolitan area have been utilized (6).

| Peak Periods: | Catalytic vehicles | Non-Catalytic vehicles |
|---|---|---|
| Hot Starts | 20% | 0% |
| Cold Starts | 50% | 30% |
| Daily Average: | | |
| Hot Starts | 10% | 0% |
| Cold Starts | 25% | 14% |

In the following sections, the data derived above is used to calculate composite emission factors for the pollutants of interest - CO, PM, $SO_4$ and Pb.

### *Carbon monoxide*

An emission factor for CO is given by the solution of the equation:

$$e = \sum_{i = n-12}^{n} c_{ipn} \, m_{in} \, v_{ips} \, z_{ipt(X)} r_{iptw\,x}$$

where:

$e$ = composite emission factor

$n$ = design year for emission calculation

$c_{ipn}$ = mean emission factor based on Federal test procedure - from Table D.1-11 in AP 42 (11,12)

$m_{in}$ = fraction of annual vehicle travel (item (3 ) above)

$v_{ips}$ = speed correction factor from Table D.1-23 (11,12)

$z_{ipt}$ = temperature correction factor from Table D.1-25 (11,12) (1.124 for non-catalyst and 1.75 from catalyst)

$r_{iptwx}$ = hot/cold vehicle operation factor for non-catalytic vehicles from Table D.1-25 (11,12)

$= \dfrac{w+xf(t)+(100-w-x)g(t)}{20+27f(t)+53g(t)} = 2.017$ when w =50% and x =20% catalytic hot starts

The results of the calculation for peak periods are:

1978 CO Composite Emission Factor

| Model Year | c | m | v | z | r | e |
|---|---|---|---|---|---|---|
| 1978 | 2.8 | .102 | 1.28 | 1.75 | 2.017 | 1.29 |
| 1977 | 9.9 | .159 | " | " | " | 7.11 |
| 1976 | 10.8 | .172 | " | " | " | 8.39 |
| 1975 | 11.7 | .130 | " | " | " | 6.87 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 1974 | 45.0 | .097 | 1.28 | 1.124 | 1.153 | 7.24 |
| 1973 | 47.0 | .083 | " | " | " | 6.47 |
| 1972 | 49.0 | .073 | " | " | " | 5.93 |
| 1971 | 66.0 | .058 | " | " | " | 6.35 |
| 1970 | 68.5 | .042 | " | " | " | 4.81 |
| 1969 | 80.4 | .031 | " | " | " | 4.13 |
| 1968 | 82.6 | .020 | 1.23 | " | " | 2.63 |
| pre-1968 | 99.0 | .026 | 1.21 | " | " | 4.04 |

Peak Period Emission Factor = 65.26 g/veh-mile

If a similar calculation is made for daily averages, the result is 47.3 g/veh-mile.

*Particulates and Sulfates*

Data for particulate and sulfate emission variation with vehicle model year and speed is sparse. AP 42 (11,12) presents average emission factors for all catalyst-equipped and non-catalyst vehicles. A general equation for average emission factor is:

$$e_{cp} = f_{ncv}e_{ncv} + f_{cv}e_{cv}$$

where:

$e_{cp}$ = composite emission factor for PM or $SO_4$

$f_{ncv}$ = fraction of non-catalyst vehicles (0.44 in 1978)

$f_{cv}$ = fraction of catalyst-equipped vehicles = $1-f_{ncv}$

$e_{ncv}$ = emission factor for non-catalyst vehicles from Table D.1-21 (11,12)

$e_{cv}$ = emission factor for catalyst-equipped vehicles from Table D.1-21 (2,3)

For particulates, the results are $e_{cp}$ = 0.38 g/veh-mile, while for sulfates $e_{cp}$ = 0.034 g/veh-mile.

*Lead*

Emission of lead from gasoline-powered vehicles may be conservatively estimated by assuming that:

- gasoline contains 2 grams lead per gallon
- 70% of lead appears as tailpipe emissions
- average vehicles get 14 miles per gallon

These considerations yield a non-catalyst emission rate of 0.1 g/veh-mile.

The lead requirement for catalyst equipped cars is 0.05 g/gallon. Using the other assumptions noted above, the emission rate for catalyst equipped vehicles equals 0.0025 g/veh-mile.

The overall emission rate is:

$$e_{Pb} = f_{ncv}\, e_{ncv} + f_{cv}\, e_{cv}$$

where:

$e_{Pb}$ = composite emission factor for lead
$f_{ncv}$ = fraction of non-catalyst vehicles (0.44 in 1978)
$f_{cv}$ = fraction of catalyst-equipped vehicles = $1-f_{ncv}$
$e_{ncv}$ = emission rate for non-catalyst vehicles = 0.1 g/veh-mile
$e_{cv}$ = emission rate for catalyst-equipped vehicles = 0.0025 g/veh-mile.

The overall emission rate for lead, $e_{Pb}$, equals 0.045 g/veh-mile.

*Idle Emissions*

Another important consideration is the emissions produced by vehicles using the proposed parking garage. Due to the extremely low speed nature of auto operation in a parking structure, idle emissions may be used to estimate such a condition. In a garage of the size proposed (2000-2500 spaces) a peak period idling time of 5 minutes has been assumed. This would apply to both entry and exit movements during congested conditions. It has also been assumed that an idling vehicle travels at the rate of 0.5 mph while entering and exiting or searching for a space. A rate of 0.5 mph over 5 minutes is equivalent to each vehicle traveling 0.0417 mile within the garage.

To predict idle CO emissions, the EPA provides a 1974 baseline emission rate and correction factors (11,12). Thus:

$$e_{CO} = \frac{\text{1974 baseline}}{\text{1974 factor}} \times \text{1978 factor}$$

$$= \frac{16.2 \text{ g/veh-min}}{67.5 \text{ g/veh-mile}} \times 42.7 \text{ g/veh-mile}$$

$$= 10.25 \text{ g CO/veh-minute}$$

Based on 5 min. = 51.25 g CO/vehicle

For $SO_4$, Pb and PM, idle emission factors may be calculated on a distance-traveled basis:

$0.034$ g $SO_4$/veh-mi X 0.0417 mile = 0.0014 g $SO_4$/veh

$0.38$ g PM /veh-mi X 0.0417 mile = 0.0158 g PM /veh

$0.10$ g Pb /veh-mi X 0.0417 mile = 0.0042 g Pb /veh

*Emission Factor Summary*

| Pollutant | At 15 mph | At Idle (5 min.) |
|---|---|---|
| CO | 65.26 g/veh-mile | 51.25 g/veh |
| $SO_4$ | 0.034 " | 0.0014 " |
| PM | 0.38 " | 0.0158 " |
| Pb | 0.10 " | 0.0042 " |

## Traffic Patterns

To complete the calculation of air pollutant emissions, one needs to consider existing and projected traffic volumes. The network volume assignments presented in Figures 1 and 2 are typical of the traffic information generated during project planning. The data presented are for weekday PM peak hour and Saturday peak hour traffic levels under both existing and proposed conditions. The data for proposed conditions, or "design level" data, were based on expected incremental traffic volume changes due to shopping activities on the fifth busiest weekday and the tenth busiest Saturday of the year. It is also assumed that 57% of the shopping trips are made by auto.

Traffic network assignments must be transformed to vehicular miles traveled (VMT) for emission calculations. This is accomplished by the expedient of totaling average traffic volumes over each roadway segment between intersections. The existing and "design level" results of VMT calculation are:

| | Weekday PM Peak Hour | Saturday Peak Hour |
|---|---|---|
| Existing traffic | 860 VMT | 820 VMT |
| Projected traffic | 980 VMT | 1005 VMT |

While the data given in Figures 1 and 2 are normally adequate for transportation facility planning, it is desirable to consider a wider variety of conditions in an air quality analysis. The greatest emission intensities and, consequently, the greatest health hazards are likely to occur as episodes of short duration. The relationship of air pollution episodes to health hazards is recognized in the federal promulgated air quality standards which specify measurement periods of 1, 8 and 24-hours for various pollutants at differing intensity levels.

Factors have been derived from basic traffic engineering parameters to transform the design levels into "worst case" forms compatible with episodal analysis. In general, one may transform from peak hour to 8-hour and 24-hour data with the following relations:

24-hour traffic = Average Daily Traffic (ADT)
Average peak hour = 7.25% ADT
Worst peak hour = 11% ADT
Average 8-hour = 48% ADT
Worst 8-hour = 60% ADT

The episodal concept may also be applied to the predicted modal split of 57% of the shoppers as drivers. A worst case condition of 100% drivers may be postulated. Similarly, the design trip levels for the fifth highest weekday and tenth highest Saturday may be projected to highest weekday and highest Saturday by using the sales effectiveness ratio. Figure 3 shows the case study plot of sales effectiveness ratios for the i-th highest shopping periods. These ratios predict the number of shopping trips generated in a given period compared to a "typical" or 30th highest level. The adjustment from the case study design levels to the highest or "zeroth" level are given by:

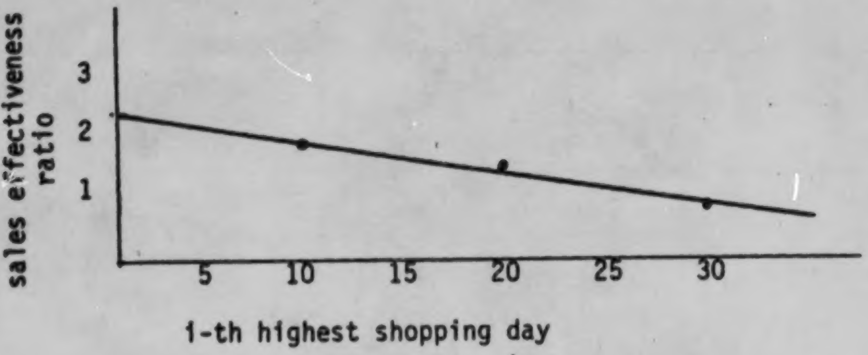

Figure 3. Sales effectiveness ratio for highest shopping days.

$$\frac{\text{highest weekday}}{\text{5th highest weekday}} = \frac{2.15}{2.00} = 1.08$$

$$\frac{\text{highest Saturday}}{\text{10th highest Saturday}} = \frac{2.15}{1.70} = 1.26$$

A set of basic traffic conditions for analysis has been defined on the basis of the preceding relationships. Each of the conditions must be evaluated at 1-hour, 8-hour and 24-hour episodal levels for both week days and Saturdays. The conditions are:

1) Existing observed traffic, a
2) Worst case existing traffic, b
   (a x $\frac{11}{7.25}$ for peak hour; a x $\frac{60}{7.25}$ for 8-hour)
3) Projected traffic generation, c
   (design level)
4) Worst case traffic generation, d
   (adjusted to "zeroth" day' c x 1.26 or 1.08)
5) Projected traffic generation - 100% driving, e
   (c/0.57 -- assume all shoppers drive autos)
6) Worst case traffic generation - 100% driving, f
   (d/0.57)

The proposed 2000 vehicle parking garage is also an important potential source of air pollutant emissions. It is analyzed for similar conditions to those designated by c - f above. However, rather than rely upon traffic network assignments, garage usage is defined by total trip-making generated by the proposed center. This is readily available data in a design study since it forms the background for derivation of network volume assignments. In this case study, the total trips are given as 1080 trips on the 5th highest weekday and 1450 trips on the 10th highest Saturday. Garage traffic movements are designated by letters g - k.

## Emission Estimates

The results of applying the previously calculated composite emission factors to the vehicular travel patterns are presented in Table 1. These data allow estimation of gross pollutant emissions during the typical periods examined by combining the appropriate conditions. Thus existing average conditons (a), or worst probable conditions (b), may be compared to projected conditions after projected implementation (a + c + g) or worst probable conditions with a normal modal split (b + d + h) or worst possible conditions with 100% drivers as an ultimate case (b + f + k).

Table 2 presents time-average emissions for 1,8, and 24 hour periods, calculated from the peak hour data in Table 1 and appropriate traffic volume adjustment factors from the Travel Patterns section. Note that Table 2 shows only the emission rates associated with Saturday afternoon peak hour traffic, since projections indicate that the highest rates are likely to occur then rather than during weekdays. By considering these values during air quality modeling, a conservative result is assured.

## AIR QUALITY MODELING

The increase in vehicles and resultant air pollution emissions as discussed in the preceeding section will, of course, yield an increase in overall air pollution concentrations within a certain distance of the proposed Shopping Center. The extent of the increase is dependent on many factors, such as: the stability of the atmosphere; horizontal wind speed and direction; the natural topography of the area as well as shape and location of man-made structures; the geometry of the area from which the increase in emissions is generated; and background or "do-nothing" case levels of the pollutants already in existence. Estimates of the pollutant concentrations which may be expected at

## TABLE 1 TOTAL EMISSIONS FOR VARIOUS TRAFFIC CONDITIONS

| | WEEKDAY | | | | | SATURDAY | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| PEAK HOUR CONDITION | VMT | CO (kg) | PM (g) | $SO_4$ (g) | Pb (g) | VMT | CO (kg) | PM (g) | $SO_4$ (g) | Pb (g) |
| (a). Existing Observed | 860 | 56 | 327 | 29 | 86 | 820 | 53 | 310 | 28 | 82 |
| (b). Worst Existing | 1290 | 84 | 490 | 44 | 129 | 1240 | 81 | 471 | 42 | 124 |
| (c). Projected Generation | 120 | 8 | 46 | 4 | 12 | 185 | 12 | 70 | 6 | 19 |
| (d). Worst Probable Generation | 130 | 9 | 49 | 4 | 13 | 233 | 15 | 86 | 8 | 23 |
| (e). Projected Generation (100% Drive) | 210 | 14 | 80 | 7 | 21 | 325 | 21 | 124 | 11 | 33 |
| (f). Worst Possible Generation (100% Drive) | 227 | 15 | 86 | 8 | 23 | 409 | 27 | 155 | 14 | 41 |
| | VEH/HR | | | | | VEH/HR | | | | |
| (g). Garage Projection | 1080 | 55 | 17 | 2 | 5 | 1450 | 74 | 23 | 2 | 6 |
| (h). Worst Probable Garage Projection | 1166 | 60 | 18 | 2 | 5 | 1827 | 94 | 30 | 3 | 8 |
| (j). Garage Projection (100% Drive) | 1895 | 97 | 30 | 3 | 8 | 2300 | 118 | 36 | 3 | 10 |
| (k). Worst Probable Projection (100% Drive) | 2046 | 105 | 32 | 3 | 9 | 2300 | 118 | 36 | 3 | 10 |

## TABLE 2 AVERAGE EMISSIONS/HOUR OF CO, PARTICULATES, $SO_4$ AND Pb FOR 1,8 AND/OR 24 HOUR TIME PERIODS

| | | TRAFFIC CONDITION | | | | | |
|---|---|---|---|---|---|---|---|
| **POLLUTANT** | | **a** | **b** | **c** | **f** | **g** | **k** |
| CO | 1 hr kg/hr | 53 | 81 | 12 | 27 | 74 | 118 |
| | 8 hr kg/hr | 44 | 55 | 10 | 18 | 61 | 80 |
| | 24 hr kg/hr | 22 | 22 | 5 | 7.4 | 22 | 29 |
| Particulates | 24 hr g/hr | 178 | 179 | 40 | 59 | 9.5 | 12 |
| $SO_4$ | 24 hr g/hr | 16 | 16 | 3.4 | 5.3 | 0.8 | 1.0 |
| Pb | 24 hr g/hr | 47 | 47 | 11 | 15.5 | 2.5 | 3.4 |

a source of air pollution and at specified distances may be made by the use of dispersion models. For this case study, the Gaussian dispersion equations described by Turner (10) were used.

## Diffusion Equations

In general, Gaussian dispersion equations were developed to describe the dispersion of an inert pollutant from a point source with a constant emission rate. The method assumes that horizontal and vertical dispersion occur in a normal distribution. The general form of the equation is:

$$C(x,y,z,H) = \frac{Q}{2\pi\sigma_y\sigma_z u}\left\{\exp\left[-\tfrac{1}{2}(y/\sigma_y)^2\right]\right\}\left\{\exp\left[-\tfrac{1}{2}\left(\frac{z+H}{\sigma_z}\right)^2\right] + \exp\left[-\tfrac{1}{2}\left(\frac{z-H}{\sigma_z}\right)^2\right]\right\} \quad (1)$$

where: $C$ = concentration of pollutant
$x,y,z$ = receptor location coordinates
$H$ = effective stack height
$Q$ = source strength
$\sigma_y, \sigma_z$ = horizontal and vertical dispersion parameters
$u$ = mean wind speed

The equation is simplified in this case study by assuming that the source and receptor are both at ground level and that all concentrations are equivalent to those along the plume centerline. Thus wind direction is not a factor, and H, y and z are set equal to zero. This point source equation then reduces to:

$$C = \frac{Q}{\pi \sigma_y \sigma_z u} \qquad (2)$$

The horizontal and vertical dispersion coefficients incorporate meterological stability and downwind distance from the source.

For area sources, a theoretical point source is modeled at some distance, d, back from the leading edge of the area source (10). The distance and horizontal dispersion coefficients are adjusted so that the plume width is equal to the width of the area source. The emission intensity at the leading edge of the area source is equal to the source strength of the area. This permits uniform application of diffusivity assumptions for both point and area sources.

The rate of pollutant dispersion is dependent upon the rate and extent of mixing in the atmosphere. The least mixing and hence the greatest concentrations of pollutant occur during stable conditons, Class F.

In this case study , wind roses detailing the frequency of 3 hour periods of Class F Stability were obtained. These figures indicate: 24.6% of all 3 hour periods experienced Class F stability; 5.5% of these F Stability periods had wind speed less than 0.5 m/sec; 5% between 0.5 and 1.5 m/sec and 14.1% had winds between 1.5 and 3 m/sec. While the majority of these F Stability periods occur at night, a significant number of them could occur during the peak traffic periods. This justifies using F Stability with a 1 m/sec wind as a reasonable approximation of the worst meteorological conditions that would be found for the air quality analyses.

In sum the background information necessary to carry out diffusion analysis, over and above gross emission intensities, is as follows:

1). The source area is a square 500 meters on a side. The actual area as shown in Fig. 2 is a rectangle approximately 400 by 600 m.

2). The area source is centered around the proposed parking facility.

3). All on-street emissions are treated as an area source contribution at ground level.

4). All parking garage emissions are treated as a point source at ground level.

5). Meteorological conditions were modeled with Class F Stability, a wind speed at 1 m/sec, and receptors always downwind from the source.

6). For study purposes, the population living in a circle of 1.5 km radius centered at the proposed parking garage is assumed to be at greatest risk from the proposed mall. This 1.5 km circular study area was divided into three 0.5 km wide concentric rings.

Furthermore, for analysis purposes it was assumed that projected concentrations at 0.5 km represent typical exposures for people living within 0.5 km of the source. The concentration of 0.75 km was considered representative of the 0.5 to 1.0 km ring and the concentration at 1.25 km represents exposures of people between 1.0 and 1.5 km from the site. Figure 4 shows an overlay of these rings on the study area.

7). Dispersion Coefficients for the appropriate study distances are (10):

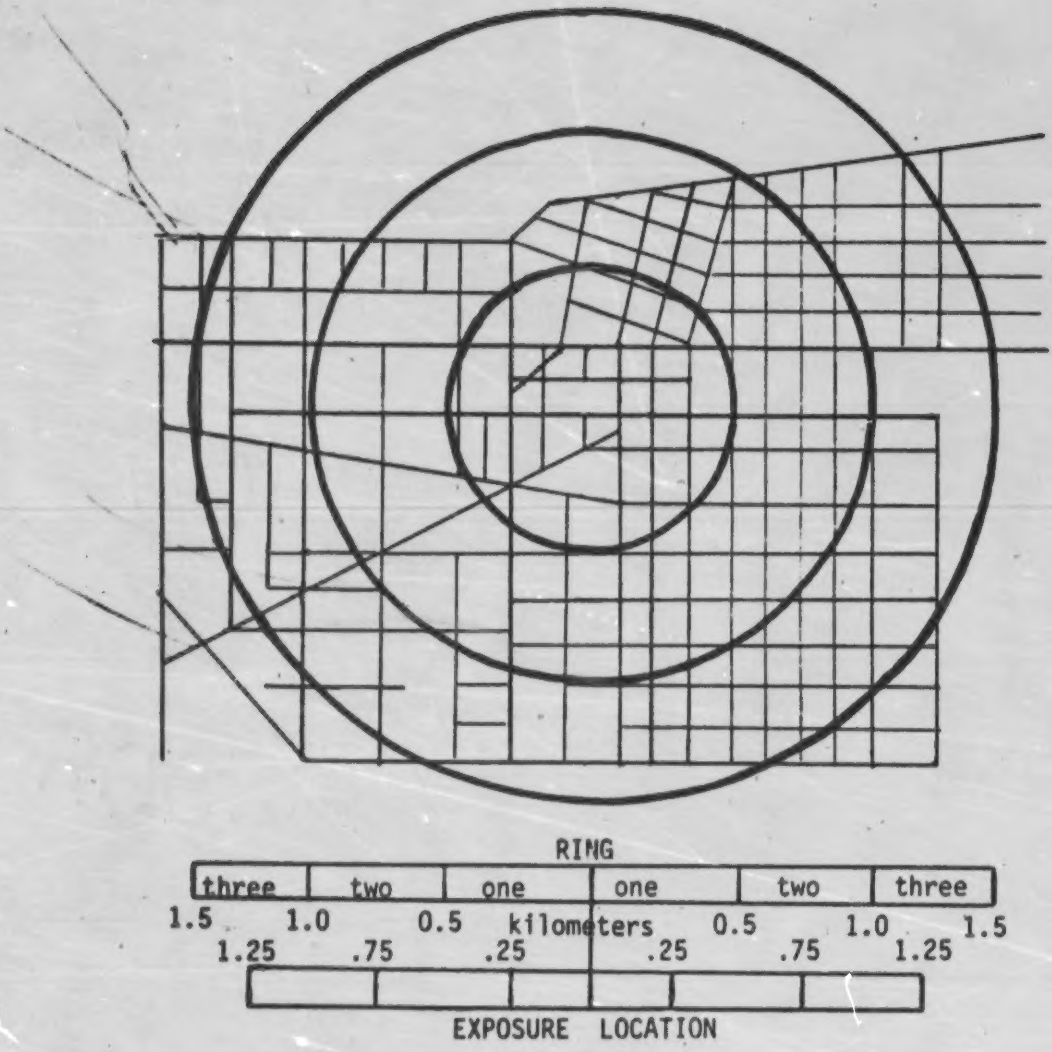

Figure 4. Population rings for the shopping center study area (rings centered on parking garage).

| Distance From Center of Source Area (x) | Virtual Distance | $\sigma_y$* | | $\sigma_z$* | |
|---|---|---|---|---|---|
| | | Point | Area | Point | Area |
| 0.5 km | 4.30 km | 18 m | 128 m | 8.4 m | 8.5 m |
| 0.75 km | 5.55 km | 26 m | 135 m | 11.5 m | 11 m |
| 1.25 km | 5.05 km | 42 m | 150 m | 16 m | 16 m |
| 1.50 km | 5.30 km | 50 m | 166 m | 18 m | 18 m |

*Class F Stability assumed

## Results of Diffusion Calculations

Concentrations for the conditions and at the distances discussed above were calculated for the following pollutants and time periods:

| | |
|---|---|
| CO | 1 hour |
| | 8 hours |
| | 24 hours |
| Particulates | 24 hours |
| $SO_4$ | 24 hours |
| Lead | 24 hours |

These time periods were chosen on the following bases:

Particulates - 24 hour standard exists

$SO_4$ - Although no standard has been set, $SO_4$ is usually associated with particulates and so the same time period was used

Pb - Damage is based on total body burden; therefore the 24 hour average exposure was considered

CO - 1 and 8 hour standards exists; 24 hour was included to enable comparison with the other 24 hour calculations

The conditions for analysis are:

(a). existing observed conditions

(b). existing highest estimated traffic volumes

(c). increment from generated traffic, 10th highest shopping day, 57% auto trips

(f). increment from generated traffic, highest shopping day, 100% auto trips

(g). increment due to garage traffic, 10th highest shopping day, 57% auto trips

(k). increment due to garage traffic, highest shopping day, 100% auto trips

Total 1 = 10th highest shopping day, 57% auto trips (a + c + g)

Total 2 = highest shopping day, 100% auto trips (b + f + k)

The value of Total 1 will reflect pollutant concentrations on a busy traffic day and poor dispersion. The difference between Total 1 and condition (a) is indicative of the incremental difference in air quality caused by the shopping center. The value of Total 2 reflects the absolute worst case (100% automobiles, poor dispersion). Results of applying the diffusion equations to the conditions above are shown in Tables 3 and 4.

Inspection of the CO data in Table 3 revealed unexpectedly high levels derived from the parking structure, modeled as a point source (g and k). It was decided, therefore, to model the garage as an area source, in the same manner as the general traffic circulation. This approach is believed to place reasonable upper and lower bounds upon the potential CO concentrations generated by the shopping center. The limits show that while the 1 hour standard of 40 mg/m$^3$ is exceeded only at the upper limit for a 0.5 km distance, the 8 hour standard of 10 mg/m$^3$ may be exceeded under the lower limit at 0.5 km and up to 1.25 km at the upper limit (Table 5). Adjustments were not made for other pollutants since the quantities emitted from the garage were relatively minor.

**TABLE 3** CARBON MONOXIDE CONCENTRATIONS FOR VARIOUS DISTANCES AND TRAFFIC CONDITIONS

Traffic Condition

| RING | DISTANCE(km) | | a | b | c | f | g | k | Total 1 a+c+g | Total 2 b+f+k | Total 1 -(a) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Concentrations in $\mu g/m^3$ | | | | | | | | |
| 1 | 0.5 | 1 hr | 4.30 | 6.58 | 0.97 | 2.19 | 43.0 | 69.0 | 48.28 | 77.77 | 43.97 |
| | | 8 hr | 3.54 | 4.48 | 0.81 | 1.50 | 35.8 | 47.0 | 40.15 | 52.98 | 36.61 |
| | | 24 hr | 1.79 | 1.80 | 0.41 | 0.60 | 12.9 | 17.0 | 15.17 | 19.43 | 13.37 |
| 2 | 0.75 | 1 hr | 3.15 | 4.82 | 0.71 | 1.61 | 21.9 | 34.9 | 25.76 | 41.33 | 22.61 |
| | | 8 hr | 2.60 | 3.28 | 0.71 | 1.10 | 18.1 | 23.8 | 21.41 | 28.18 | 18.81 |
| | | 24 hr | 1.32 | 1.32 | 0.30 | 0.44 | 6.52 | 8.62 | 8.20 | 10.37 | 6.82 |
| 3 | 1.25 | 1 hr | 1.95 | 2.99 | 0.44 | 0.49 | 9.8 | 15.1 | 12.19 | 22,59 | 10.24 |
| | | 8 hr | 1.60 | 2.03 | 0.37 | 0.68 | 8.0 | 10.6 | 9.97 | 13.31 | 8.37 |
| | | 24 hr | 0.81 | 0.82 | 0.18 | 0,27 | 2.90 | 3.8 | 3.89 | 4.43 | 3.08 |
| | 1.50 | 1 hr | 1.78 | 2,73 | 0.40 | 0.91 | 7.2 | 11.6 | 9.38 | 15.24 | 7.60 |
| | | 8 hr | 1.46 | 1.85 | 0.34 | 0.62 | 6.00 | 7.9 | 7.80 | 10.37 | 6.34 |
| | | 24 hr | 0.75 | 0.75 | 0.17 | 0.25 | 2.17 | 5.72 | 3,08 | 6.72 | 2.34 |

## TABLE 4 TWENTY-FOUR HOUR CONCENTRATIONS OF LEAD, SULFATE AND PARTICULATE MATTER FOR VARIOUS DISTANCES AND TRAFFIC CONDITIONS

| RING | DISTANCE (km) | | Traffic Conditions a | b | c | f | q | k | Total 1 a+c+q | Total 2 b+f+k | Total 1 -(a) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Concentrations in $\mu g/m^3$ | | | | | | | | |
| 1 | 0.5 | Pb | 1.71 | 1.71 | 0.40 | 0.57 | 0.66 | 0.90 | 2.77 | 3.17 | 1.06 |
| | | $SO_4$ | 1.3 | 1.3 | 0.28 | 0.43 | 0.47 | 0.58 | 2.05 | 2.31 | 0.75 |
| | | PM | 14.5 | 14.5 | 3.3 | 4.8 | 5.5 | 7.1 | 23.3 | 26.4 | 8.8 |
| 2 | 0.75 | Pb | 1.26 | 1.26 | 0.29 | 0.42 | 0.33 | 0.45 | 1.89 | 2.12 | 0.63 |
| | | $SO_4$ | 0.96 | 0.95 | 0.21 | 0.32 | 0.24 | 0.30 | 1.41 | 1.57 | 0.45 |
| | | PM | 10.6 | 10.7 | 2.4 | 3.5 | 2.8 | 3.6 | 15.8 | 17.8 | 5.2 |
| 3 | 1.24 | Pb | 0.78 | 0.78 | 0.18 | 0.26 | 0.15 | 0.20 | 1.11 | 1.24 | 0.33 |
| | | $SO_4$ | 0.60 | 0.58 | 0.13 | 0.19 | 0.10 | 0.13 | 0.83 | 0.90 | 0.23 |
| | | PM | 6.6 | 6.6 | 1.5 | 2.2 | 1.2 | 1.6 | 9.3 | 10.4 | 2.7 |
| | 1.50 | Pb | 0.72 | 0.71 | 0.17 | 0.23 | 0.11 | 0.15 | 0.99 | 1.09 | 0.27 |
| | | $SO_4$ | 0.54 | 0.54 | 0.12 | 0.18 | 0.18 | 0.10 | 0.74 | 0.82 | 0.20 |
| | | PM | 6.0 | 6.0 | 1.4 | 2.0 | 0.9 | 1.2 | 8.3 | 9.2 | 2.3 |

**TABLE 5** CARBON MONOXIDE CONCENTRATIONS FOR PARKING GARAGE MODELLED AS AN AREA SOURCE

($mg/m^3$)

| Distance | Time | Condition a | Condition c | Condition g* | Condition g | Lower Limit | Upper Limit † |
|---|---|---|---|---|---|---|---|
| 0.5 km | 1 hour | 4.30 | 0.97 | 5.98 | 43.0 | 11.25 | 48.28 |
| | 8 hour | 3.54 | 0.81 | 4.95 | 35.8 | 9.30 | 40.15 |
| 0.75 km | 1 hour | 3.15 | 0.71 | 4.38 | 21.9 | 8.24 | 25.76 |
| | 8 hour | 2.60 | 0.71 | 3.62 | 18.1 | 6.93 | 21.41 |
| 1.25 km | 1 hour | 1.95 | 0.44 | 2.71 | 9.80 | 5.10 | 12.19 |
| | 8 hour | 1.60 | 0.37 | 2.24 | 8.0 | 4.21 | 9.97 |
| 1.5 km | 1 hour | 1.78 | 0.40 | 2.47 | 7.2 | 4.65 | 9.38 |
| | 8 hour | 1.46 | 0.34 | 2.04 | 6.0 | 3.8 | 7.80 |

g* Models garage as 0.5 km square area source - (a) + (c) + (g*) = lower limit.

† Models garage as a point source - (a) + (c) + (g) = Total 1

## RECEPTORS

Certain populations are known to be more susceptible to the adverse effects of pollutants than other population groups. For example, people with respiratory problems are generally more sensitive to ozone pollution than normally healthy individuals. Young children are sensitive to all pollutants because of their high metabolic rate. Though we cannot with any degree of confidence state the exact type and extent of physiological responses (and associated health effects) corresponding to different levels of concentration of a specific pollutant, we can identify and enumerate those groups potentially at risk from pollutants generated by a new installation. The types, numbers, and locations of the susceptible groups found within the study area are presented below.

### Sources of Data and Enumeration Procedures

Information on the total population, number of persons over 62, and children less than 5 in the study area was gathered from the 1970 census. Census blocks were assigned to the study rings according to geographic area. Population per ring was thus obtained by summing over all blocks assigned to that ring.

The population of persons less than 5 years of age is not available in block data, but was obtained indirectly by the formula:

$$S_i = \sum_{j=1}^{n} E_{ij} \cdot TP_{ij} \cdot CT_{ik}$$

where:

$S_i$ = total persons less than 5 years of age located within ring i.

$E_{ij}$ = proportion of total population in block j, located in ring i.

$TP_{ij}$ = total population for block j, located in ring i

$CT_{ik}$ = proportion of those under 18 who are less than 5 residing in census tract k within which block j is located

The above formula assumes that the proportion of all persons under 18 years of age who are less than 5 years old is the same for both the census block and census tract within which that census block is located. This may not be the case as the actual proportion for each block is likely to be somewhat greater or less than the overall proportion for the census tract, depending on the geographic distribution of 5 year olds. However, the above formula probably gives a good approximation of the number of persons less than 5 who are located within each of the rings in 1970.

Information on enrollments in elementary schools was obtained from the local school district. The State Department of Public Health provided information on long-term care facilites and hospitals in the area. Other local groups were able to provide enrollment data for nurseries, day care centers and senior citizen centers.

Note that the information based on cnesus data is for 1970. Obviously, none of those persons under 5 years of age in 1970 are under 5 in 1976. The number of elderly (over 62) may have decreased or increased depending on the healthiness of those over 62 and the number of persons 57 to 62 years of age in 1970. Total population probably did not change very much in the area from 1970 to 1976 because very little open land was available for development in 1970. Applying general population trends, we might expect the number of persons over 62 years of age to have slightly increased and the number of persons less than 5 years of age to have slightly decreased from 1970 to 1976. The population of hypersusceptibles considered to be at risk from the pollutants is summarized in Table 6.

## TABLE 6 SOME POPULATION GROUPS AT HIGH RISK TO POLLUTANT EXPOSURES BY DISTANCE FROM THE SOURCE AREA AND POLLUTANT

| SUSCEPTIBLE GROUP | RING | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Total Population | 4959 | 12530 | 13227 |
| Children under 5 years old | 155 | 724 | 912 |
| Day Care Centers/Nurseries | 72 | 146 | 190 |
| Elementary Schools | 0 | 672 | 1006 |
| Hospital Beds | 44 | 0 | 56 |
| Long-Term Care Beds | 110 | 60 | 5 |
| Population over 62 years old | 995 | 2757 | 1658 |
| Asthmatics (total for the whole area) | | 920 | |
| Persons with emphysema (total for the whole area) | | 600 | |
| Bronchitics | | 950 | |
| Persons with Heart Disease (total for the whole area) | | 716 | |

## HEALTH EFFECTS AND POPULATIONS AT RISK

Although no specific dose/response relationships have been developed for most pollutants, the physiological effects of exposures to various ranges of pollutants are generally known.

### Lead

Lead (Pb) has long been recognized as a health hazard, traditionally associated with central nervous system effects. As a metabolic poison, lead causes affected cells to use oxygen inefficiently. Lead is regarded as a poison which can accumulate in the body, and the contribution of inhaled lead to the body burden is not insubstantial even at low exposure (i.e. 1.5 $\mu g/m^3$). (3) Children are particularly susceptible to lead effects. There is evidence that low-level lead exposures in children are capable of producing behavioral disorders, learning disability and changes in heme synthesis. A number of studies have suggested that lead emitted from automobiles is a health hazard to children. One study has found a relationship between elevated blood lead levels (a measure of lead body burden) and traffic volume in children 1 to 5 years old residing near heavy traffic density. (1)

The area near the proposed shopping center contains a significant number of population groups considered at risk from lead. Almost 1800 children under 5 years old reside in the 1.5 km-radius area around the shopping center. Lead levels on the 10th worst shopping day of the year with 57% of the shoppers driving to the shopping center may average 1 to 3 $\mu g$ $Pb/m^3$ of air depending on the distance from the center. (Tables 4 and 6)

The Environmental Health Resource Center in Illinois has recommended a standard of 1.5 $\mu g$ of $lead/m^3$ for a 24-hour average. If emissions estimated in Table 4 were to prevail on weekdays, children spending their days attending elementary schools, day care centers and nurseries may be exposed to lead levels well above 1.5 μg/m$^3$. Almost 700 children attend K-8 schools only 0.75 km from the center, where they may be exposed to 24 hour average levels of over 2 μg/m$^3$. Of additional concern are 70 toddlers who attend a day care center at a distance of 0.5 km from the center when Pb levels may reach 3 μg/m$^3$.

## Sulfates and Particulates

Forms of sulfur oxides, including sulfates ($SO_4$), are highly irritating to the lining membranes of the respiratory tract. The irritant effect may cause narrowing of the air passages, impairing the clearance of inert particles from the lung and efficient transfer of respiratory gases. Considering these effects, population groups of particular concern include the elderly, young children, and those with pre-existing diseases of the heart and pre-existing lung diseases such as asthma, chronic bronchitis and emphysema.

The current federal standard for particulates is 260 μg/m$^3$ for a 24 hour average and 75 μg/m$^3$ for an annual average. Table 4 shows that under worst case conditions, only 23.3 μg/m$^3$ or 10% of the 24 hour standard could be generated by the shopping center.

There are no current federal, state or local standards for $SO_4$. The $SO_4$ and particulate concentrations generated by the development are below levels that have been correlated with adverse health affects (2,8,13). However, the impact of other sources of these pollutants have not been included in the air quality analysis. It is possible, therefore, that the development could have an adverse impact to certain people living in the area.

Based on the 1970 census and a high risk population estimation technique, (4) there are approximately 920 asthmatics, 950 bronchitics, and 600 people with emphysema living within 1.5 km of the proposed shopping center who could be affected by the increased $SO_4$ and particulates shown on Table 4. In addition, Table 6 shows 1781 children 5 years of age, 408 nursery school children and 1678 elementary school children who could be harmed by incremental increases in sulfates and particulates.

## Carbon Monoxide

Groups most susceptible to the toxic effects of CO are infants, and persons with cardiovascular disease, anemia, lung disease, and/or increased metabolic rate (9).

Exposure to carbon monoxide (CO) results in reduction of the oxygen supply to various organs. Those afflicted with heart disease are of particular concern in CO exposure studies. Heart attack victims hospitalized in areas of relatively high CO levels are found to experience a greater mortality rate than those at hospitals in areas of lower CO levels. Recent studies of angina patients exposed to CO (37-100 ppm) showed that after exposure, the mean duration of exercise befor onset of pain was significantly shortened compared to that obtained as a result of breathing clean air (15).

The U. S. EPA has established CO standards of 40 $mg/m^3$ for 1 hour and 10 $mg/m^3$ averaged over 8 hours, not to be exceeded more than once per year. These standards are based primarily upon impairment of time-interval discrimination, psychomotor functions, and visual discriminization, and upon physiologic stress on heart patients. As shown in Table 3, the one hour standard may be exceeded at up to 0.75 km from the shopping center under the worst possible conditions. Similarly, the 8 hour standard may be exceeded at up to 1.5 km.

The effect of CO upon humans is reflected by the level of carboxyhemoglobin (COHb) present in the blood. In Table 7, some example

## TABLE 7 PREDICTED CARBOXYHEMOGLOBIN LEVELS IN NON-SMOKERS FOR VARIOUS DISTANCES AND TRAFFIC CONDITIONS*

| Distance from Center of Study Area, (km) | Traffic Condition | | |
|---|---|---|---|
| | (a) | Total 1 | Total 2 |
| | % COHb for 8-Hour Exposure | | |
| 0.5 | ≤ 1.0 | 4.6 | 6.0 |
| 0.75 | < 1.0 | 2.2 | 3.5 |
| 1.25 | < 1.0 | 1.7 | 1.9 |
| 1.50 | < 1.0 | 1.2 | 1.5 |

* For smokers, levels may be higher by 4% COHb

COHb levels for non-smokers exposed to CO concentrations for the existing and proposed traffic conditions are shown. These levels have been calculated from the CFK equation of Peterson and Stewart (7).

## SUMMARY AND CONCLUSIONS

The results of this study project a decrease in overall air quality within 1.5 km of the new shopping mall. This change in air quality could have an adverse impact on a total of 30,700 residents, of which 10,000 persons are identified as hypersensitive to air pollution.

Potential violations of ambient air quality standards for carbon monoxide have been identified for distances up to 1.25 km from the center. Evidence for concern over lead, sulfate and particulate levels has also been raised. Given the fact that numerous simplifying assumptions and data approximations have been utilized, the results still highlight the need for detailed study of any project of this type and magnitude. Air quality considerations clearly must form an essential part of project planning. Emphasis should be placed upon minimizing dependence upon the automobile for trip making, reduction of vehicular idling times, and attention to the proximity of hypersusceptible population groups during site selection.

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# EVALUATION OF PLANNING ALTERNATIVES:
# COAL HYDROGENATION EVALUATION*

Janet A. Holden and Richard A. Wadden

## BACKGROUND

The most abundant energy resource in the United States is coal. Identified deposits that are recoverable with present technology are calculated to be 12-19 times greater than U. S. petroleum reserves (35). In 1974, the 600 million tons of coal produced accounted for only 18% of total U. S. energy consumption. However, the use of coal for energy is projected to increase substantially over the next few decades and production could reach over 1 billion tons by 1985 (10). Because of the demand for low sulfur, environmentally acceptable fuels, conversion of coal to synthetic fuels is being investigated. The technology for coal liquefaction and gasification has been demonstrated on a commercial scale in Germany, England and South Africa. Environmental measurements from these plants, however, were either not made or are not available.

At the present time, no commercial scale liquefaction or gasification plants exist in the United States. There are some plants in the planning stages and a number of pilot plants either are operating or have been demonstrated. Evaluation of the potential environmental effects of coal processing depends on data from these pilot plants and from bench scale reactors.

Commercially feasible gasification plant sizes will typically reduce in excess of 20,000 tons/day of coal to products and wastes (2,24, 33). This quantity of coal is roughly equivalent to that fed to

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* Part of this presentation was excerpted from Wadden, R. A., "Coal Hydrogenation and Environmental Health", Environ. Health Perspect. 14:201-206, 1976.

3-700 MW electrical power plants. Because of the large amounts of material being processed, the potential exists for environmental discharge of considerable quantities of hazardous materials. These include hydrogen cyanide, phenols, cresols, carbonyl and hydrogen sulfides, ammonia, mercaptans, thiocyanides, aniline, arsenic, trace metals, and various polycyclic hydrocarbons (5,7,12,17,22,29,31,36,37).

Sources of possible pollution from hydrogenation plants can generally be identified with three categories: the nature of the raw material, the design of the processing scheme, and the way in which the process is actually operated (37). Coal has a very complex, heterocyclic structure, which must be broken down to produce desirable products (Fig. 1). A given coal conversion process will destroy many of these bonds. Thiophene, methylpyridine, hydroxynaphthalene and benzo(a)-pyrene are only a few of the compounds which could reasonably be expected to be produced. However, the cleavage of these cyclic linkages, and the chemical form of the resultant compounds, are difficult to analyze and predict. As is indicated in Tables 1 (27,28) and 2 (14), coal also contains significant quantities of hazardous metals and considerable amounts of sulfur and nitrogen. For example, arsenic, beryllium, mercury, lead, and vanadium have all been identified as materials with properties toxic to man (39). Functional groups in coal include the methoxy, hydroxyl, carbonyl, and carboxyl groups. These can lead to the formation of contaminants and hazards such as phenols, metal carbonyls, aromatic nitrogen and sulfur compounds (18).

Many different coal hydrogenation processes have been attempted or projected (3). Each needs to be analyzed specifically for the types of wastes produced and how they are distributed to water, air and soil. However, some general observations can be made about the nature of the conversion process. Most hydrogenation processes require energy addition at elevated temperatures and pressures in order to break down the coal. Table 3 indicates the atomic H/C ratios of

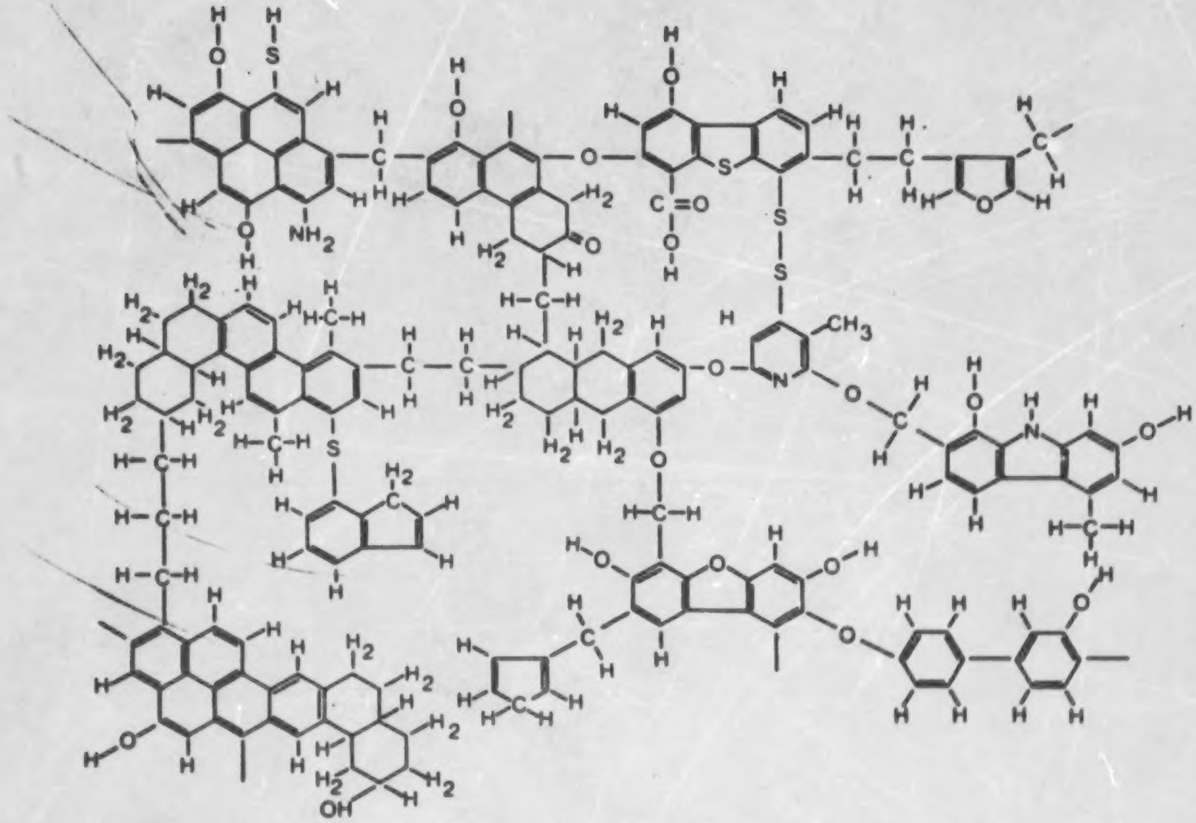

FIGURE 1. A representative bituminous coal structure. Based on the structure given by Hill and Lyon (*15*).

Table 1. Some Trace Elements in Coal and Coal Gasifier Gas (37)

| Element | Concentration, lb of trace element/1000 tons of coal | |
|---|---|---|
| | Coal[a] | Coal gasifier gas[b] |
| As | 28.0 | 27.7 |
| B | 204.0 | 97.3 |
| Be | 3.2 | 0.1 |
| Cd | 5.0 | |
| Co | 19.1 | |
| Cr | 27.5 | |
| Cu | 30.3 | |
| F | 122.0 | 121.0 |
| Hg | 0.4 | 0.4 |
| Mn | 98.8 | 6.8 |
| Mo | 15.1 | 13.7 |
| Ni | 42.1 | |
| Pb | 69.6 | 25.2 |
| Sb | 2.5 | 1.9 |
| Se | 4.2 | |
| Sn | 9.6 | |
| V | 65.4 | |
| Zr | 145.0 | |

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[a] Data of Ruch et al (27).

[b] Calculated from data of Sather et al (28).

Table 2. Chemical Analysis of Typical Illinois No. 6 Coal.[a]

| | Concentration, tons/1000 tons dry coal |
|---|---|
| Carbon | 707 |
| Hydrogen | 54 |
| Nitrogen | 10 |
| Sulfur | 50 |
| Oxygen | 80 |
| Ash | 99 |

[a] Data of Helfinstine et al (14); Table from Wadden (37).

Table 3. Hydrogen/Carbon Atomic Ratios in Typical Fuels (37).

| Fuel | Atomic ratio H/C |
|---|---|
| Coal (Illinois No. 6) | 0.86 |
| Heating oil | |
| Residual (No. 6) | 1.55 |
| Distillate (No. 2) | 1.79 |
| Naphtha | 2.20 |
| LPG | 2.65 |
| Natural gas | 3.40 |
| Pure methane | 4.00 |

various raw materials and finished products. The necessary energy is ordinarily derived from oxidizing a portion of the incoming coal. Hydrogen is supplied either through direct or indirect utilization of the hydrogen atoms in water or through use of a hydrogen donor solvent.

In a typical coal hydrogenation plant, the coal is first ground, dried and preheated and then fed to a coal gasification unit. In the gasification step, the coal is reacted with steam to yield carbon monoxide and hydrogen. The hydrogen then reacts further with the coal to yield gas with a low heating value (approximately 200 Btu/ft$^3$) and char. In order to obtain pipeline gas (approximately 950 Btu/ft$^3$) further conversion is required. In the shift reaction, carbon monoxide is reacted with more steam over a catalyst to yield hydrogen. In the methanation step, carbon monoxide reacts with the hydrogen to yield a hydrogen-rich high Btu product gas. The major reactions are summarized below:

$$\mathrm{C(coal)} + \mathrm{H_2O} \rightarrow \mathrm{CO} + \mathrm{H_2}$$

$$\mathrm{C(coal)} + \mathrm{H_2} \rightarrow \underset{\text{(low Btu value)}}{\mathrm{CH_4} + \text{char}}$$

Gasification Reaction

$$\mathrm{CO} + \mathrm{H_2O(Steam)} \xrightarrow[\text{cat.}]{} \mathrm{CO_2} + \mathrm{H_2}$$

Shift Reaction

$$\mathrm{CO} + 3\mathrm{H_2} \xrightarrow[\text{cat.}]{} \underset{(\approx 950\ \text{Btu/ft}^3)}{\mathrm{CH_4} + \mathrm{H_2O}}$$

Methanation Reaction

The product gas is cleaned of $H_2S$ and $NH_3$ and the hydrogen is removed and recycled to the reaction step. The liquid products are distilled to yield light and heavy fuel oil products. Sulfur and ammonia are also formed in marketable quantities.

Temperatures of 700-1000°F and pressures up to 1500 psig are needed for hydrogenation. The specific operating conditions will determine what gaseous and solid byproducts are formed, as well as the product ratios of gas/liquid.

Coal gasification yields enormous quantities of waste products and heat. The products are formed directly as gases, solids or liquids and are cleaned out of the waste streams by means of water treatment or such waste removal processes as scrubbers, cyclones, and bag houses. Each treatment process, of course, yields final waste products.

Waste char and ash, which require disposal, amount to 20-30% of the raw coal (2,34). Some phenolic material is likely to be disposed of with the waste ash and char and has the potential to be leached out into water systems (17). A variety of catalysts, including compounds containing iron, zinc, aluminum, copper, and cobalt, are used in coal hydrogenation (6,9). Perhaps of greatest concern are the types utilized for methanation in the gasification process. Nickel, Co, Fe, Ru, Pd, Os, and Pt have all been suggested as possible catalysts. The presence of nickel carbonyl (a suspected carcinogen) (1) has been reported in methanizer product gas (25). Since temperatures $<525^{\circ}F$ encourage the formation of $Ni(CO)_4$, and reduced conversion of CO to $CH_4$, careful temperature control is necessary for process as well as health reasons. In certain industrial settings, Pt and Pd have been implicated in the occurrence of an asthma-like disease, although their existence in the gas phase in significant concentrations is problematical (19,26).

Under hydrogenation conditions the trace elements in coal represent potential sources of toxic arsine, borane, hydrofluoric acid, organo mercury and selenide emissions. Coal contains several chelating structures that may have various effects on the mobilization and release of many metals (18). Due to the toxicity, and in some cases, carcinogenicity of some organometallic coal conversion products, these substances must be considered and their possible emissions evaluated.

Heavy oil and tars produced by hydrogenation may comprise a quantity of 30,000-200,000 lb/1000 tons coal (33,36). At present, these materials are not completely characterized. A partial analysis of some of the constituents contained in the tar from a gasification pilot plant is shown in Table 4 (12). It has been suggested that the heavy oils can be used (and consequently controlled) by recycling them to the main hydrogenation reactor for further reduction and/or by using them to slurry the incoming coal (34). However, there are some data that suggest that the naphthalenic fraction of recycle streams is difficult to break down (7). There is also evidence that some higher molecular weight materials may not be completely degraded (16,38). In addition, product liquids may have higher aromatic contents than naturally occurring hydrocarbons with the same boiling point characteristics (30). Since many cyclic hydrocarbons have been identified as hazardous to man, the potential of these liquids for causing adverse effects on the central nervous system, liver, kidneys, and skin, as well as their possible carcinogenicity, needs to be analyzed.

Only one study of the actual health hazards of coal hydrogenation is presently available (17,31,32,38). A group of 359 workers was examined for skin cancer over a period of 5 years at a liquefaction plant designed to process 300 tons/day of incoming coal. Of this group, 10 men developed skin cancer. Exposure varied from several months to 23 years, but all significant lesions were found in workers with less than 10 years of exposure. The reported incidence was 16-37 times the incidence of skin cancer expected in the chemical industry (32). Analyses of the liquid materials produced in the plant identified a number of carcinogenic materials. An increase in carcinogenicity with increasing liquid boiling point, usually with respect to liquids with boiling points >260°C, was observed in laboratory animals (38). This relationship was particularly evident for the oil, produced in the plant, which was recycled to slurry the

Table 4. Some Tar Constituents in the Synthane Coal-to-Gas Process.[a]

| | Concentration, Vol % |
|---|---|
| Naphthalene | 11.6 |
| Fluorenes | 9.6 |
| Three-ring aromatics | 13.8 |
| Phenols | 4.0 |
| N-Heterocyclics | 10.8 |

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[a] Data of Forney et al (12); Table from Wadden (37).

incoming coal. In addition, air measurements in working areas revealed benzo(a)pyrene concentrations often in excess of 50 μg/1000m$^3$ with a peak value of 18,000 μg/1000m$^3$ (17). Benzo(a)pyrene has been identified as being strongly carcinogenic (23). These concentrations are in contrast to typical urban levels of 3-6 μg/1000m$^3$ (23).

Health statistics on occupational groups in other coal conversion operations, such as coke ovens and coal-tar processing, have shown significantly higher lung cancer rates relative to groups without such occupational exposures (8,20,21,23). In particular, coke oven workers appear to have 2.5 times the incidence of lung cancer as other steel workers (20). The environment on top of a coke oven is, of course, different from that likely to occur in hydrogenation plants which process material continuously. There is no assurance that the same incidence of cancer or any other disease will occur. However, close control of environmental discharges is strongly suggested in light of these occupational observations and the hydrogenation plant data cited above.

## EXAMPLE OF EVALUATION METHODS

The following example shows how the framework discussed above was applied to evaluate an Environmental Analysis Report (EAR) (4) for a proposed demonstration coal hydrogenation plant. In order to accurately determine the environmental impact of this plant, a minimum of three steps were needed:

1) Identification and quantification of amounts of hazardous materials which might be discharged from the plant.
2) Estimation of possible dispersion of these materials in air and water.
3) Identification and enumeration of populations who might be exposed to discharges and determination of their exposures.

In order to accomplish these three steps the following information was needed from the EAR:

1) The amounts and composition of the plant's input and output streams.
2) Process flow diagrams with temperatures, pressures, flow rates and compositions for each stream. The analysis of such flow diagrams identifies the locations where the greatest degree of control is required.
3) The amounts of hazardous materials produced and the types and efficiency levels of control equipment.
4) Historical meteorological and hydrological data for the surrounding area.
5) Historical air, water and soil pollutant values for the surrounding areas, as well as types and location of other major pollutant sources.
6) Population densities and locations, as well as enumeration of special subgroups, such as children and the elderly.

## Plant Input and Output Streams

Analyses of the types of raw coal projected for use in the hydrogenation plant were not included in the EAR. As a result, no determinations could be made of possible trace element discharge to air, water or soil. In addition, data on sulfur levels of incoming coal were conflicting in different sections of the EAR. Consequently, the discharge of sulfur to the environment could not be accurately determined.

The other major feedstock is water. The EAR did include extensive information about the quality and quantity of the water to be used. This information was obtained from state agencies as well as by independent testing. The testing samples were taken from only three locations, one time each in September, November and December. This sampling protocol did not give an accurate representation of water quality changes throughout the year. For example, it ignored the problems of summer low flows and increased agricultural runoff carrying fertilizers and pesticides during spring rains.

Other feedstocks to be used were (in the order listed in the EAR) air, nitrogen, lime, alum, $H_2SO_4$, caustic, chlorine. The amounts of each of these substances and their uses was unclear. Their potential chemical reactivities as well as intermediate and ultimate forms when released into the environment were not discussed.

The composition of the final products was not well defined. Specifically, the amounts of $H_2$ in the product gases and the energy values for all products were not given. There was no mention of ammonia in any of the lists of final products and yet, in a general description of the process, the production of saleable ammonia was reported. Whether or not ammonia will be produced, during which steps, and wastes or byproducts formed as a result of production needed to be clarified.

## Process Description

The EAR contained a very general and confusing description of the processes to be used in the plant. Only vague references to "required pressures" or "high enough temperatures" were included. The composition of the streams between the different units were not specified and no flow rates or quantities were given. Figure 2, from the EAR, shows the level of information provided. The temperature and pressure figures were obtained from the text.

This lack of information makes it impossible to estimate the amounts, types and sources of emissions or to accurately predict the accident and/or explosion potential of the different process units in the plant.

Start up and shut down procedures produce completely different temperature, pressure, and emission levels at all points in the plant than do normal operating conditions (37). Neither the usual method of

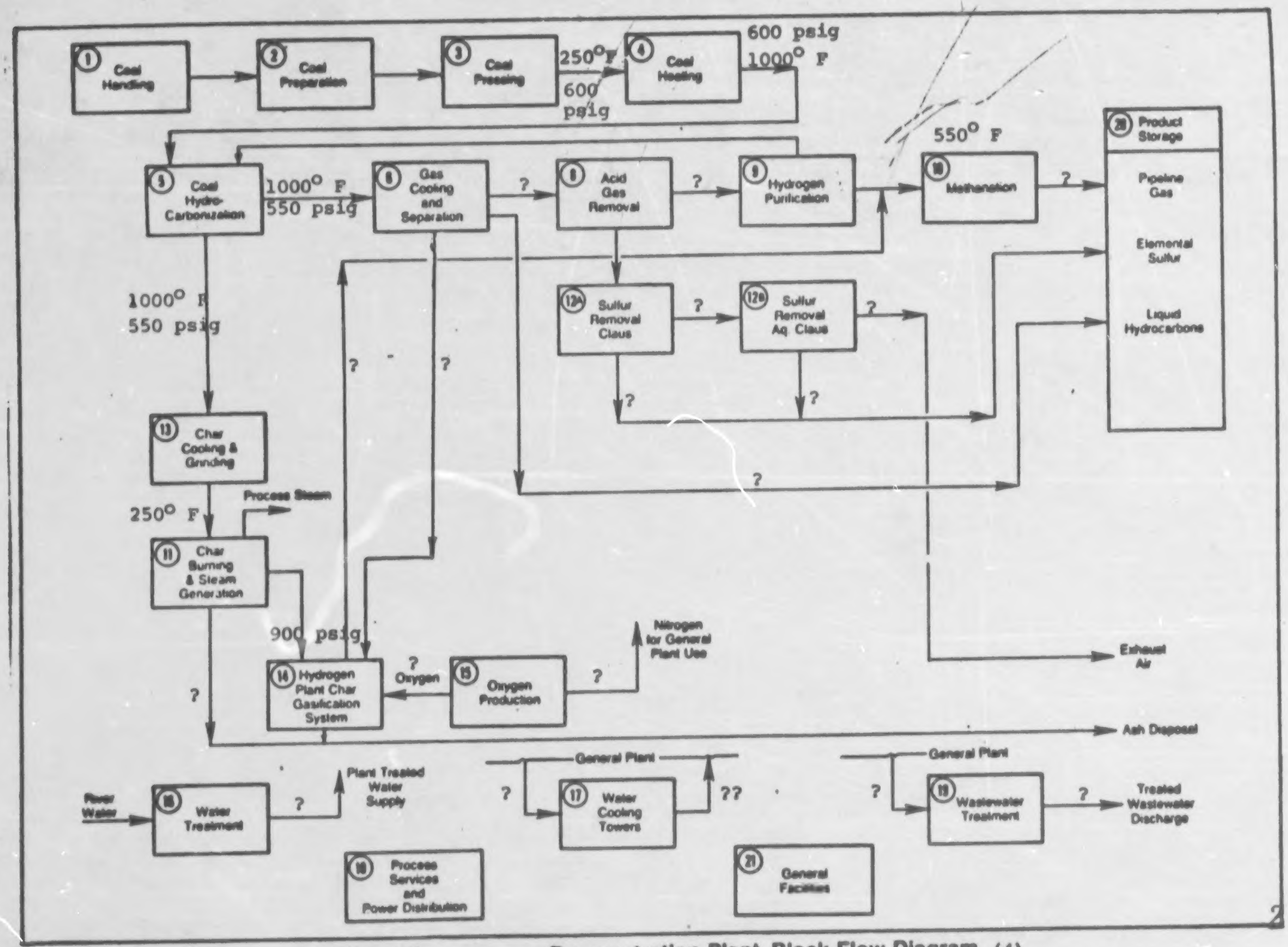

Figure 2 Demonstration Plant Block Flow Diagram (4)

operation nor any procedures or equipment to be used during start up, emergency or routine shut down are discussed in the report.

Quantitation of Hazardous Wastes and Control Equipment Design

No design or efficiency information for wastewater and air pollution control equipment was included in the EAR except for a general discussion of the Claus plant for sulfur removal. Scrubbers and cyclones were mentioned in various process description steps but without design specifications. Based on our estimates, the control equipment described for the Claus plant emissions will not remove the particulates and $SO_2$ to the required best available control technology levels.

For the whole plant, only particulates, $SO_2$ and COS, and non-methane hydrocarbon emissions in air and total amounts of wastewater were given. None of the assumptions used to calculate these emissions were indicated.

The results of diffusion modeling for $SO_2$ and particulates were reported in the EAR as:

$SO_2$ - maximum annual arithmetic mean concentration increase = 5.0 - 6.0 $\mu g/m^3$

Particulates - maximum annual geometric mean concentration increase = 5.0 $\mu g/m^3$

None of the assumptions used for this modeling were given. The area that will be affected by this concentration increase was also not stated. The Air Quality Control Region in which the plant will be located has been designated as Class II. Therefore, the allowable increase in particulate concentration from all sources is limited to 10 $\mu g/m^3$ and the increase for $SO_2$ is limited to 15 $\mu g/m^3$ (11). The preliminary modeling results, although unexplained and unsupported, indicated that emissions from the plant will use one-half of the allowable particulate increment increase and one-third of the allowable $SO_2$ increment increase. It appears that higher control efficiencies than those assumed in the modeling will probably be required. These concentration increase values are especially important in light of the fact that this is a demonstration plant, approximately one-fourth of the size of the proposed commercial scale plant.

The amounts and types of solid waste to be produced by the plant were not addressed in the report. Brief mention was made of the solids from the waste treatment units and an estimated 400-450 tons/day of flyash. No characterizations of these potentially very hazardous materials were attempted. When we carried out detailed elemental balances, significant discrepancies between plant input and output data were encountered.

### *Sulfur*

A crude mass balance for sulfur was considered essential in view of the potential adverse health effects of sulfur emissions. The average of the sulfur content listed in the feedstocks was used, a value of 3.3% S by weight. The mass balance is given in Table 5. The fate of the unaccounted sulfur (1.3T/D) was unclear from the report. A more basic question, however, is how the $SO_2$ and COS emissions were estimated from the range of sulfur input levels. Furthermore, there was no mention of the amount of anhydrous sodium sulfate produced as a by-product of the sulfur removal process. The possibility of $H_2S$, $SO_2$ , or sulfate aerosol emissions from the aqueous Claus plant were also not addressed. The amount of sulfur possibly present in the wastewater was not estimated.

### *Carbon*

The proposed plant will convert the highly complex coal molecular structure into simpler hydrocarbon fuels. Its main concept involves hydrogenation of the organic fraction of coal with extensive severing of carbon-carbon and carbon-heteroatom bonds to form simple hydrocarbons. Therefore, carbon content conservation is a significant

Table 5. Sulfur Balance

| Material | T/D | Sulfur Fraction | Sulfur T/D |
|---|---|---|---|
| Input | | | |
| Coal | 3850 | 0.033 | 127.0 |
| Output | | | |
| Liquid Sulfur Product | 122 | 1.000 | 122.0 |
| $SO_2$ from Claus Plant | 7.2 | 0.500 | 3.6 |
| COS from Hydrogen Plant | 0.2 | 0.505 | 0.1 |
| Total Output | | | 125.7 |
| Unaccounted Sulfur | | | 1.3 |

measure of the plant efficiency. That portion of the carbon not transformed to product represents a potential source of air and water contamination. A carbon balance is shown in Table 6. As is indicated in the table almost 1700 T/D carbon are unaccounted for.

### *Other elements*

Some of the varying amounts of trace elements which may be present in coal are presented in Table 2. The fate of these and any other trace materials such as those used as catalysts were not considered in the EAR.

### *Mass and energy balances*

Due to the amount of missing information, complete and comprehensible mass and energy balances were not possible. It is clear from the sulfur and carbon balances alone that a vast quantity of material is unaccounted for in the project description materials. Most of this is made up of water but a coal balance shows that some of this material is also missing. (See Table 7 ).

Water. Attempts to carry out a water balance for the plant were not successful. The only quantities specified were 4.6 MGD input, 0.61 MGD wastewater output and 4 MGD consumed for boiler feed and process water. Since we estimated that the hydrogen needs of the plant would be satisfied by only 0.45 MGD of process water, there remains about 3.5 MGD unaccounted for. If the water evaporates from the cooling towers, it would seem that such a large quantity may create severe problems such as ground fogs and local weather alterations. The combination of large quantities of water vapor with particulates, carbon dioxide, sulfur dioxide, and sulfates is of serious concern with respect to acid rain.

Some of the possible elements to be discharged in the wastewater output were indicated in the EAR, but were not quantitated. Projected

**Table 6.** **Carbon Balance**

| **Material** | **T/D** | **Carbon Fraction** | **Carbon T/D** |
|---|---|---|---|
| **Input** | | | |
| Coal | 3850 | 0.71 | 2,733.0 |
| **Output** | | | |
| Heavy Fuel Oil | 550 | 0.89 | 489.5 |
| Light Hydrocarbons | 165 | 0.84 | 138.6 |
| LPG | 96 | 0.82 | 77.9 |
| SNG | 425 | 0.78 | 331.5 |
| Air Emissions | | | |
| $C_2$ | 0.3 | 0.80 | 0.2 |
| $C_3$ | 0.6 | 0.82 | 0.5 |
| **Total Output** | | | **1,038.2** |
| **Unaccounted Carbon** | | | **1,694.8** |

**Table 7.** **Coal Balance**

| | T/D |
|---|---|
| **Coal Input** | |
| Moisture (9.0%) | 346 |
| Ash (9.3%) | 359 |
| Sulfur (3.3%) | 127 |
| Carbon (71.0%) | 2733 |
| Other (7.3%) | 285 |
| Total In | 3850 |
| **Coal Output** | |
| Flyash (250 - 400 T/D) | 359. |
| Sulfur | 126. |
| Moisture (8% Removed) | 308. |
| Particulates | 1.8 |
| Carbon in Products | 1037.5 |
| Carbon for $H_2$ Production | 1257. |
| Carbon in Air Emissions | 0.8 |
| Carbon for Plant Energy | 437.7 |
| Solids in Wastewater | 6.4 |
| Total Out | 3534.2 |
| Difference between In and Out | 315.8 |

water effluent quality in terms of physical/chemical parameters was listed, but the method and assumptions used to develop the projections were not.

Energy and heat. It was not possible to construct an accurate energy balance from the information in the EAR. Development of such a balance would require accurate heating values of all products, a typical mix of coals to be used and heating values of those coals, energy requirements of all processes in the proposed plant, and a complete listing of all temperatures and pressures throughout the plant.

## Meteorological and Hydrological Data

In order to accurately estimate the effects of air and water contaminants emitted by any new pollutant source, the existing conditions in the area must be evaluated.

The EAR presented and analyzed meteorological data for the past 20 years for the area surrounding the proposed plant. These data included temperature, windspeed and direction, amount of precipitation, and the percentage of time that inversions, fog and smog were present. In addition, the microclimate for the immediate plant site was analyzed using information on topography, vegetation and hydrology. The tornado risk at the site was also calculated. If the appropriate emission data had been provided, this historical information would have been useful for dispersion calculations. Other information needed for truly accurate modeling of pollutant levels would be the percent time each stability class is experienced in the area.

Extensive hydrological data were also provided. This includes drainage of surface water and surface water availability, regional groundwater patterns and the effect of strip mining on the groundwater in the immediate vicinity of the proposed plant. Again, more information is needed to put these data to use.

## Ambient Pollutant Values and Other Pollutant Sources in the Area

### *Air*

Air pollutant levels for the Air Quality Control Region in which the plant would be located were listed in the EAR. National Air Surveillance Network data and measurements taken by the local power company were utilized. However, no information was given as to sampling times or frequency. Air quality sampling at the plant site was carried out for a one-week period. The information gathered during that time is of limited value since it cannot be representative of seasonal fluctuations, nor does it provide enough of a base from which to calculate annual average pollutant levels. The air quality data provided does indicate that the Air Quality Control Region does not meet the National Air Quality Standards for particulates, $SO_2$, ozone, and CO at the present time. The prevailing winds in the area of the proposed plant site are predominantly from the south in the summer months. A major fossil fuel power plant which burns high sulfur coal is located eight miles to the south of the proposed plant. During the winter, the winds are from the northwest. More monitoring data is needed to determine the effects of a major metropolitan area located to the northwest of the site. This area is heavily industrialized, the largest industries being primary and fabricated metals, chemicals, and coal and oil production. Meaningful dispersion estimates could not be made due to the lack of emission and design information.

The need for characterization of the quantities and types of emissions from the plant, as a first step in the assessment of the potential threat to human health, is strongly indicated in light of the following information provided in the EAR:

1. Prevailing winds from the direction of the major point and area sources.
2. An annual average inversion frequency of 36%.
3. An annual average fog frequency of 10%.

### *Water*

Water quality data were reported from two public agencies as well as some on-site testing. The testing was conducted one day in each of the months of September, November and December and so it cannot represent water quality changes throughout the year nor give yearly averages. No indication of sampling times or frequencies for the agency data was given. The data indicated that the river to be used as the receiving water for the proposed plant effluent already exceeds the State Water Quality Standards for phenol, iron, and manganese. Organic contaminants were found in fish from the river in one instance. It is unclear how much sampling was done for organic contaminants in aquatic organisms. In addition, ground water at the site, already disturbed by strip mining, has been found to exceed the standard for total dissoved solids and iron.

The river serves as the major source of potable water for a town located downstream from the proposed plant. In addition, the river is stated to be a major source of recreation, chiefly boating and fishing, for the county. It is impossible to estimate the effect on the river quality from available data for the proposed plant. The already existing standard violations and possibility of agricultural and strip mined land drainage indicate that very strict controls will be needed on the water effluent in order to protect the health of those using this resource.

### Population Data

Extensive compilations of the population data in the counties surrounding the proposed plant were included in the report. Some breakdown on a countywide basis was made with regard to age, sex and race. The purpose of including much of these data was unclear. Locations of schools, day care and nursing homes were not determined. The number of hospitals in the major communities was listed, but the locations were not given. No estimates for the number of chronically ill were made.

Correlation of the location of highest population densities and pollutant dispersion estimates was not done. The location of the most susceptible groups, children and elderly, was done on too broad a basis for population exposures to be made even if accurate pollutant dispersion estimates were possible. The possiblity of health effects to the residents in the vicinity of the plant were summarized as follows: "Contaminant concentrations are not likely to create any adverse impact outside of the site area because of their projected low levels." (4) However, this statement contradicted earlier statements in the report that projections of these concentrations cannot be made. Occupational health was not addressed in the report.

## Conclusion

The Environmental Analysis Report discussed here was prepared as a preliminary document by which the decision is made whether or not to prepare a full Environmental Impact Statement. Our evaluation provided a method to point out those areas which were lacking and what information is still needed for a comprehensive and worthwhile Impact Statement. Our analysis was necessarily incomplete. However, our approach did supply the basis for asking the right questions so that a rational evaluation could be carried out.

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